AMERICAN JOURNAL

OF

DISEASES OF CHILDREN

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VOLUME III

1912

PUBLISHERS

AMERICAN MEDICAL ASSOCIATION

CHICAGO, ILL.

MU

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BACTERIOLOGICAL

American Journal of Diseases of Children

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STUDIES ON INFANT NUTRITION

UNDER THE DIRECTION OF L. EMMETT HOLT, M.D., AND P. A. LEVENE, M.D.

II. The Hard or Casein Curds in Infants' Stools *

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The casein curds in infant stools have been the subject of much discussion and controversy in recent years. Considerable disagreement exists in regard to both their chemical composition and the mechanism of their formation. All writers have been unanimous, however, in attributing to the curds much clinical significance, and in using them as a basis for choosing, or altering, the diet.

The older authorities regarded them as casein residues, appearing in the stools as the result of insufficient protein digestion and absorption. In recent years very emphatic protest against this view was expressed by Keller and Czerny.[1] According to their view, these curds were composed principally of a conglomeration of fat and soaps. Still more recently Biedert's[2] original view on the curds was revived, particularly by American writers. Selter,[3] Southworth and Schloss,[4] and Talbot[5] made chemical analyses of the curds and on the basis of his results reached the conviction that the principal component of these masses was undigested casein. The cause of their presence in the stools lay, according to these writers, in the disturbed protein digestion. Against this view there immediately arose opposition, led principally by the school of Finkelstein. L. Meyer and Leopold[6] did not deny the fact that the curds contained protein material, but, however, regarded the source of it not the casein of the ingested milk, but the intestinal secretions.

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*From the Laboratories of the Rockefeller Institute for Medical Research, and the Babies' Hospital, New York.

1. Czerny and Keller: Des Kindes Ernährung, etc., 1906, part 2, 17.

2. Biedert: Jahrb. f. Kinderh., 1881, xvii, 251; Kinderernährung im Säuglingsalter, etc., 1905, ed. 5.

3. Selter: Versamml. Deut. Naturforsch. u. Aerzte in Stuttgart in 1907, 177.

4. Southworth and Schloss: Arch. Pediat., 1909, xxvi, 4, 241.

5. Talbot: Boston Med. and Surg. Jour., 1908, clviii, No. 24, p. 905; 1909, clx, No. 1, p. 13; 1910, clxii, No. 5, 134; Arch. Pediat., 1909, xxvi, No. 12, p. 919.

6. Meyer, L. F., and Leopold: Arch. Ped., 1909, xxvi, No. 10, p. 773; 1910, xxvii, No. 2, p. 126.

The cause of the appearance of the curds lay, according to the view of these writers, not in insufficient protein metabolism, but in faults of the carbohydrate assimilation and was brought about by excessive carbohydrate intake. These authors maintained that the appearance of the curds in the stools could be abated when the sugar intake was lowered.

Thus all writers are in agreement on one point, namely, that the presence of the hard curds in the stools is an indication of some fault of digestion, and that the health of the infant is in danger if the curds persist in the stools. The disagreements relate only to the interpretation of the nature of the disturbance and to the mode of treatment advocated by individual writers.

A mere superficial scrutiny of the literature on the subject reveals the fact that even the gravity of the symptom has never been firmly established; it was an assumption based on speculative considerations. Taking this assumption for granted, the individual observers applied their efforts in search for a method of treatment by which the symptom could be abated. The interpretation of the pathogenesis of the symptom was then determined by the success or failure of a certain mode of treatment. Thus, if the removal, complete or partial, of carbohydrate from the diet led to the disappearance of the curds, the theory was advanced that the disturbance of carbohydrate digestion was the basic cause of this symptom. Arguments of this nature can scarcely be regarded as very convincing. On the other hand, it was never clearly demonstrated that there existed a definite form of indigestion which invariably resulted in the curd formation in the stools, and still less evidence existed in favor of the assumption that the nutrition of the infants was affected severely in the periods when the stools contained the curds in abundance.

New experimental inquiry was needed in order to obtain reliable information on all these questions, and the present work was stimulated by such considerations.

Thus it is natural that the work should fall into two parts, one dealing with the relation of the curds to the state of digestion, and the second to that of nutrition. Information relating to the first part of the problem was sought on the grounds of the following reasoning: If the curds represent the remnants of undigested food, then their composition should be determined by the nature of the digestive insufficiency. Thus, if the curds consist principally of one component, of protein, of fat, or of carbohydrate, the conclusion would follow that there existed an insufficiency in the digestive capacity for that food-stuff. If that were so, one should also find that the composition of the entire stool showed a preponderance of that same component. On the other hand, should the curds have exactly the composition of normal stools differing only in the water content, the assumption may be justified that their formation

is conditioned by irregularities of peristalsis. Finally the mass of the stools may, under some forms of indigestion, acquire such peculiarities of composition as would lead to the separation of one of its constituents in solid form. Thus, the accumulation of calcium salts may result in separation of lime-soaps; unusually high acidity may bring about precipitation of nucleins. Under such conditions one should expect the curds to have a composition distinctly different from that of the remainder of the stools, but would expect also a definite constant composition of the stool as a requirement for the curd formation.

From these considerations it is evident that it is not sufficient to limit the chemical investigation to the composition of the curds except when it is desired to establish the relationship between the state of digestion and the curd formation. On the contrary, it is very essential to supplement the analysis of the curds by that of the total stools. Furthermore, the insufficiency of the digestive organs may be essentially quantitative in its nature. That is, it may occur only after the intake of a certain constituent of the daily diet has overstepped a certain limit. Thus in order to be able to take into account all the factors engaged in the curd formation it is necessary to possess the knowledge of the chemical composition of the food, of the curds, and of the total stool in which they occur.

The data furnished by such analyses suffice to bring to light the bearing of any gastro-intestinal disturbance on curd formation, but they cannot help to interpret the influence of the curd formation on the state of general nutrition. It has been stated that there exists no experimental evidence in support of the view that the curds in infants' stools are indicative of some serious disturbance of nutrition. And yet the importance of the entire problem of curd formation depends on the answer to this question. In order to approach the solution of this problem the analyses of the food, of the curds, and of the total stools were supplemented by that of the urine, so that at least in regard to the nitrogen the exact daily balance could be established. Taking into account, in addition to this, the curve of the daily gain in weight, one can obtain fair information as to the general state of nutrition during the periods when the stools showed the greatest number of curds.

One other possibility needs to be analyzed before the discussion on the significance of the curds is exhausted, namely: Assuming that the curds represent a part of the ingested food which escaped digestion, one can easily recognize that the gravity of this condition would depend on the proportion of the food which escapes in that form. If the loss is considerable then normal nutrition could be maintained only by a corresponding increase in the daily diet. This information can be obtained by an estimation of the quantities of food elements contained in all the

curds present in a twenty-four-hour sample of the stools. Naturally the curds have to be cleansed very carefully from the adhering feces. Such analyses were carried out in the course of the present investigation. They are recorded in Table 2.

Before passing to the report on the results of the present investigation it seems necessary to describe the general character of the curds, which are referred to in the present communication. It has been pointed out by several writers that much of the controversy in regard to the significance of the curds has resulted from the fact that writers were not sufficiently explicit in describing the appearance and the physical properties of the curds, and while some were dealing actually with fat-curds, other writers had under their observation curds of an entirely different composition. The curds referred to in the present paper possessed great similarity in their physical properties, though they differed considerably in their form.

Physical Character of the Curds.—Very commonly they consisted of rather hard, round or oval, not infrequently bean-shaped masses. Often they presented masses tapering at one end, or flattened into thin plates. The surface was generally smooth. The size varied from small fragments to masses more than an inch in length. The weight of such curds reached 2 gm. In several cases the combined curds obtained from a twenty-four-hour sample of feces (three or four large and two or three smaller masses) weighed between 7 and 8 gm. The surface was frequently stained deep yellow, while the inner part was usually white or cream-colored, more rarely yellow. In a few instances they presented a shining surface, persistent even after careful washing. Very often more or less numerous small fragments of the same material as the hard curds were found in the stools, either with or without the larger masses, possibly resulting from disintegration of the latter. These hard curds have the common characteristic of being more or less tough and resistant to pressure, and can thus be distinguished from the so-called fat-curds. The latter are generally soft and small in size.

On one occasion there was obtained a smooth, oval, moderately hard mass about 1¼ inches in length, which became liquid during the drying on a water-bath. On analysis it was found to contain over 80 per cent. of fat and between 1 and 2 per cent. nitrogen. Macroscopically it differed from the typical hard curds only in yielding more readily to pressure and in showing a smoother texture. The feces of the same child contained for a long period previous to that day the characteristic casein curds.

Chemical Composition.—Results of complete analysis of the curds are recorded by Selter[3] and by Talbot.[5] The total number of samples analyzed was eight, one by Selter and seven by Talbot. In regard to the protein content, there was an approximate uniformity in all the

Composition of Curds
Per Cent. of Dried Weight of Curds
Food Composition •

Number	Name	Date	Food Composition *							Composition of Curds Per Cent. of Dried Weight of Curds				Description of Stools Containing Curds
			Protein, Per Cent.	Fat, Per Cent.	Carbohydrate, Per Cent.	Nitrogen, gm.	Fat, gm.	Carbohydrate, gm.	Total Number Calories	Nitrogen	Protein	Fat	Ash	
		Nov. 1910												
1.	E. A.	5	1.75	...	5	3.02	....	54.0	334	9.8	61.2	20.0	...	Pasty; granular, yellow-green.
2.	A. P.	9	1.2	1.4	6	2.02	14.7	63.0	465	10.5	65.6	22.5	4.8	Pasty; granular, green-yellow, sour.
3.	S. R.	26	1.4	1.6	5.7	2.35	16.8	59.9	487	10.6	66.2	25.1	...	Loose; granular, green.
		Dec.												
4.	W. E.	7	2.3	.9	2.6	3.31	8.1	23.4	304	10.6	66.2	22.3	4.8	Loose-watery; granular, green.
5.	H. C.	7	1.8	.9	5	2.72	8.5	47.2	376	11.3	70.5	....	6.5	Pasty; granular, with mucus, green-yellow.
6.	H. S.	7	1.8	.9	2.25	3.03	9.5	23.6	307	10.6	66.2	19.0	4.8	Pasty; smooth-granular, green-yellow.
7.	P. C.	12	1.6	1.8	7.8	2.42	17.0	73.6	544	9.9	61.8	29.5	5.0	Pasty; smooth, pale yellow-green, sour.
8.	I. W.	16	1.4	1.6	5.7	2.35	16.8	59.9	487	9.5	59.3	....	...	Pasty; smooth-granular, yellow-green.
9.	T. S.	20	1.75	2.0	5	3.03	21.6	54.0	538	9.0	56.2	22.0	4.8	Pasty; smooth, yellow becoming green-brown, sour.
10.	T. S.	21	1.75	2.0	5	3.03	21.6	54.0	538	8.8	54.9	22.0	4.8	Loose-pasty; granular, green and yellow.
11.	T. B.	30	1.4	1.6	5	2.35	16.8	52.5	459	10.1	63.1	....	4.8	Pasty; granular, yellow becoming green.
		Mar. 1911												
12.	C. R.	7-8	2.05	...	2.65	3.45	....	27.8	251	11.4	71.2	18.8	...	Loose; smooth, with mucus, yellow-brown becoming green.
13.	J. H.	13-14								9.4	58.7	22.0	...	Pasty; smooth, pale gray-yellow, slightly green.
14.	J. H.	14-15	2.0	2.9	5	3.54	32.1	55.3	663	10.1	63.1	....	...	Loose-pasty; smooth, pale yellow, slightly green.
15.	J. H.	17-21	2.0	1.8	5	3.54	19.9	55.3	549	9.4	58.7	....	...	Loose-pasty; mostly smooth, green-yellow and yellow-gray.
16.	J. H.	21-22	2.2	2.0	5	4.07	23.1	57.7	609	8.8	54.9	....	...	Loose-pasty; smooth, yellow becoming white-gray and pale green.

No. *Food

1. Fat-free milk 50 per cent., lime-water 5 per cent., barley-water 45 per cent., lactose 5 per cent. Food boiled.
2. Milk 35 per cent., lime-water 5 per cent., barley-water 60 per cent., lactose 5 per cent., and 12 c.c. malt-soup extract.
3. Milk 40 per cent., lime-water 5 per cent., barley-water 55 per cent., lactose 5 per cent., and 8 c.c. malt-soup extract.
4. Mixture of full milk, fat-free milk, 5 per cent. lime-water and barley-water making milk fat 0.9 per cent., lactose 2.6 per cent.
5. Skimmed milk 50 per cent., lime-water 5 per cent., barley-water 45 per cent., lactose 5 per cent.
6. Skimmed milk 50 per cent., lime-water 5 per cent., barley-water 45 per cent., lactose 2.25 per cent.
7. Milk 45 per cent., lime-water 5 per cent., barley-water 50 per cent., lactose 5 per cent., 30 c.c. malt-soup extract.
8. Milk 40 per cent., lime-water 5 per cent., barley-water 55 per cent., lactose 5 per cent., 8 c.c. malt-soup extract.
9. Milk 50 per cent., lime-water 5 per cent., barley-water 45 per cent., lactose 5 per cent.
10. Same as in No. 9.
11. Milk 40 per cent., lime-water 5 per cent., barley-water 55 per cent., lactose 5 per cent.
12. Fat-free milk 55 per cent., lime-water 5 per cent., water 35 per cent., lactose 2.65 per cent.

13 and 14. Mixture full milk, fat-free milk, lime-water 5 per cent., and barley-water making milk fat 1.8 per cent., lactose 5 per cent., olive-oil 12 c.c.

15. Same as in Nos. 13 and 14, with olive-oil omitted.
16. Similar mixture to that in preceding numbers, but making milk fat 2 per cent, lactose 5 per cent.

curds. Regarding the fat content Talbot recorded a considerable variation in different samples. According to this observer, the fat content of the curds was determined by that of the food. In the course of the present observations more than thirty samples of curds were analyzed. Great care was taken to free the curds from adhering feces. This was accomplished by shaking them with water. The wash waters were removed by decantation, and the curds dried and used for analysis. The chemical analysis of these masses revealed a similarity in their composition which cannot be regarded as accidental. It is clearly seen from the tables that the protein, fat and ash content fluctuated within very narrow limits.

On the other hand, the composition of the stools was very variable, the nitrogen content fluctuating between 3, 4 and 5 per cent., only in one instance reaching 5.5 per cent., and in another 6 per cent.; the proportion of fat varied from 26 to 48 per cent., the ash between 20 and 31 per cent. The total acidity of the feces and the content of volatile acids also presented great variations.

Thus it is evident that the hard curds are composed differently than the mass of the stools and not infrequently contain more than 75 per cent. of protein. On this ground they cannot be regarded as masses of feces compressed into the peculiar formations owing to some irregularities of intestinal motility. They therefore represent either remnants of food that escaped normal digestion, or a normal component of the feces separated out of the mass of the stools owing to some peculiarity of the composition of the latter. The fact that the chemical nature of the feces that contained the curds differed very markedly lends more support to the first of the two hypotheses, namely, that the curds represent remnants of undigested food, principally protein.

This naturally leads the inquiry into the causes of this partial indigestion. It has to be admitted that a very definite answer to this question cannot be given notwithstanding all the data furnished by the present work.

The great variations in the chemical composition of the stools which show the presence of curds argue against the assumption that there exists one definite form of indigestion which brings about the curd formation. To the same conclusion one is led by the analysis of the influence of diet on the appearance of curds. It is clearly seen from Table 1 how varied the diet may be which renders possible the formation of curds in the feces. And still the impression is gained that the protein intake is a very significant factor in bringing about this symptom. Particularly the metabolism experiment on Daniel L. seems to furnish evidence in support of this view. This patient was under observation for considerable time and his diet was altered several times. It was observed that the curds

TABLE 2.—COMPOSITION OF HARD CURDS (FROM TWENTY-FOUR HOURS' FECES)

Food Composition *	Output, Per Cent. of Dried Weight of Curds	

TABLE 2.—COMPOSITION OF HARD CURDS (FROM TWENTY-FOUR HOURS' FECES)

Number	Name	Date	Food Composition *							Dried Weight of Stools, Including Curds, gm.	Dried Weight of Curds, gm.	Output. Per Cent. of Dried Weight of Curds				Description of Stools Containing Curds
			Protein, Per Cent.	Fat, Per Cent.	Carbohydrate, Per Cent.	Nitrogen, gm.	Fat, gm.	Carbohydrate, gm.	Total Number Calories			Nitrogen	Protein	Fat	Ash	
		Dec. 1910														
1.	D. L.	6-7	2.1	1.8	5	4.58	24.6	68.3	686	12.13	1.00	9.0	56.2	23.3	....	Pasty; constipated, smooth, yellow-gray
2.	D. L.	7-8								17.82	3.60	10.1	63.1	23.3	....	Loose-pasty; smooth, yellow, green flakes.
3.	D. L.	12-13	1.8	1.2	5	3.94	16.4	68.3	582	11.20	2.85	10.2	63.7	24.0	4.6	Pasty; smooth, yellow becoming green.
4.	D. L.	13-14								9.88	1.00	10.4	65.0	24.0	4.6	Pasty; constipated, smooth, yellow becoming gray-brown.
5.	D. L.	15-16								11.18	2.80	11.0	68.7	24.0	4.6	Pasty; part granular, yellow becoming green, part smooth, brown.
6.	D. L.	17-18								6.02	.70†	1.3	8.1	81.5	14.7	Pasty; constipated, smooth, yellow becoming gray-brown, with mucus, small hard curds and one large fat lump.
7.	D. L.	18-19								6.30	.45	10.9	68.1	24.0	4.6	Pasty; smooth, yellow becoming gray-brown.
8.	R. M.	14-15	1.8	1.2	5	3.33	13.9	57.8	492	8.69	.30	11.5	71.9	19.6	6.3	Pasty; constipated, smooth, yellow becoming gray-brown.
9.	R. M.	16-17								13.84	.90	10.9	68.1	19.6	6.3	Pasty; constipated, smooth, yellow becoming gray-brown.
10.	R. M.	18-19								5.58	.10	10.9	68.1	19.6	6.3	Pasty; constipated, smooth, yellow becoming gray-brown and slightly green.
11.	J. D.	7-8	2.1	1.8	5	3.53	18.9	52.5	527	10.63	3.15	10.5	65.6	30.0	4.0	Pasty; finely granular, smooth, yellow becoming green, sour.
12.	J. D.	9-10								10.33	2.90	10.0	62.5	30.0	4.0	Pasty; partly granular, smooth, yellow becoming green, partly smooth, yellow becoming white.
13.	J. D.	12-13	1.8	1.2	5	3.33	13.9	57.8	492	8.92	3.65	10.8	67.5	30.0	4.0	Pasty; smooth, yellow becoming gray-brown.
14.	J. D.	14-15								6.22	1.30	11.5	71.9	30.0	4.0	Pasty; mostly granular, yellow becoming green, partly smooth, yellow becoming white and green.
15.	J. D.	15-16								6.23	1.55	10.5	65.6	30.0	4.0	Pasty; smooth, green and yellow, becoming brown and green.
16.	J. D.	17-18								10.98	4.65	11.1	69.3	30.0	4.0	Pasty; part smooth, yellow becoming brown, white and green, part granular, green and sour.
17.	J. D.	18-19								6.67	2.55	10.6	66.2	30.0	4.0	Pasty; part smooth, yellow becoming green and brown, part granular and green.

*Food.—Nos. 1, 2, 11, and 12.—Mixture full milk, fat-free milk, 5 per cent. lime-water and water making milk fat 1.8 per cent.; lactose 5 per cent.

In all other numbers, mixture full milk, fat-free milk, 5 per cent. lime-water and water making milk fat 1.2 per cent.; lactose 5 per cent.

†Fat lump only, small hard curds not separated from stool.

appeared in the highest number and more continually when the percentage of protein intake reached the highest values. The only period when the stools were free from curds was when both the fat and protein intake were low and the carbohydrate intake comparatively high. The variation in fat intake alone remained without influence on the curd formation.

TABLE 3.—COMPOSITION OF STOOLS CONTAINING HARD CURDS—FECES WITHOUT THE CURDS

Name	Date	Total Nitrogen, gm.	Per Cent. of Dried Weight of Feces					Per Cent. Soap Fat of Total Fat	C.c. N/10 NaOH Equivalent			
			Nitrogen	Protein	Fat	Soap Fat	Ash		Total Acidity of Feces	Volatile Acids of Feces	100 gm. Dried Weight Feces: Total Acidity	100 gm. Dried Weight Feces: Acids Volatile
	Dec. 1910											
D. L.	6-7	.592	4.9	30.6	29.7	19.2	26.4	64.6	45.2	103.5	406	930
D. L.	7-8	.782	5.5	34.4	30.4	11.4	22.6	85.2	85.2	112.9	599	794
D. L.	12-13	.278	3.4	21.2	45.0	25.3	19.8	56.3	38.9	44.8	804	1,010
D. L.	13-14	.531	6.0	37.5	36.9	17.5	25.9	42.3	17.7	172.9	199	1,947
D. L.	15-16	.407	4.8	30.0	36.2	16.3	25.2	45.2	6.8	47.6	354	2,386
D. L.	17-18	.226	4.3	26.8	33.6	16.6	24.2	49.3	3.4	47.3	121	1,771
D. L.	18-19	.282	4.8	30.0	36.9	17.5	25.9	42.3	4.9	88.3	83	1,510
R. M.	14-15	.376	4.5	28.1	33.6	25.3	29.4	75.3	8.4	179.9	100	2,144
R. M.	16-17	.618	4.8	30.0	33.6	25.3	29.4	75.3	4.1	36.8	52	437
R. M.	18-19	.285	5.2	32.5	33.6	25.3	29.4	75.3	5.7	43.7	104	798
J. D.	7-8	.288	3.8	23.8	46.2	26.3	20.9	56.9	33.6	77.8	448	1,040
J. D.	9-10	.311	4.2	26.2	43.0	31.2	21.2	72.6	31.4	92.7	422	1,248
J. D.	12-13	.230	4.4	27.5	48.8	38.7	21.4	79.3	10.2	83.3	332	2,724
J. D.	14-15	.198	4.0	25.0	37.7	19.7	25.1	52.2	12.0	65.2	569	3,697
J. D.	15-16	.236	5.1	31.8	36.3	24.5	19.3	67.5	20.2	58.1	580	1,909
J. D.	17-18	.263	4.3	26.8	45.1	33.9	22.8	75.3	4.7	30.0	236	1,736
J. D.	18-19	.201	4.7	29.4	38.1	26.6	20.3	69.8	7.7	68.8	354	3,533
	March, 1911											
J. H.	13-14	.141	4.5	28.1	26.8	15.8	26.4	59.0				
J. H.	14-15	.164	4.3	26.8	32.4	19.2	31.2	59.3				
J. H.	17-18	.094	3.8	23.8	48.6	35.9	25.3	73.9				
J. H.	18-19	.170	4.1	25.6	31.5	13.2	22.5	41.9				
J. H.	19-20	.136	3.9	24.4	28.0	8.1	23.0	23.9				
J. H.	20-21	.245	3.8	23.8	35.4	16.7	28.3	47.2	17.5	44.8	270	693
J. H.	21-22	.247	3.3	20.6	48.3	5.0	24.2	10.4	23.1	72.4	311	976
J. H.	22-23	.123	3.7	23.1	39.8	15.9	20.6	39.9				

Thus it seems probable that under a variety of conditions, part of the protein intake escapes being digested and is removed by the stools in form of hard curds. The real mechanism of the curd formation is not easy to interpret for the reason that normally protein enters the intestinal canal from the stomach in a state of solution, and even if some of it enters in solid form the proteolytic enzymes of the pancreas are capable of bringing it into solution. If the protein is of the nature of a nuclein or paranuclein, as, for instance, casein, the alkalies present in the intestinal tract should be capable of effecting its solution. There is undoubt-

edly a sufficient supply of proteolytic enzymes in the intestinal canal even under the conditions favorable to abundant curd formation, for the reason that often only a comparatively small part of the ingested protein appears in the stools in that form, the greatest part having apparently undergone normal digestion. Thus there must be some factors at play, which prevent the enzymes from penetrating the curd. No definite explanation of their nature can be offered at the present moment.

TABLE 4.—COMPOSITION OF STOOLS CONTAINING HARD CURDS—FECES INCLUDING THE CURDS

Name	Date	Feces. Dried Wt. in gm.		Total Nitrogen, gm.	Total Fat, gm.	Total Ash, gm.	Per Cent. of Dried Weight of Feces			
		With Curds	Without Curds				Nitrogen	Protein	Fat	Ash
	Dec. 1910									
D. L.	6-7	12.13	11.13	.682	3.53	2.99	5.6	35.0	29.3	24.6
D. L.	7-8	17.82	14.22	1.146	5.16	3.61	6.4	40.0	28.9	20.3
D. L.	12-13	11.20	8.35	.567	4.47	1.79	5.1	31.9	39.9	16.0
D. L.	13-14	9.88	8.88	.635	3.52	2.35	6.4	40.0	35.7	23.8
D. L.	15-16	11.18	8.38	.719	3.72	2.24	6.4	40.0	33.3	20.0
D. L.	17-18	6.02	5.32*	.235	2.37	1.39	3.9	24.4	39.3	23.0
D. L.	18-19	6.30	5.85	.331	2.27	1.53	5.3	33.1	36.1	24.4
R. M.	14-15	8.69	8.39	.411	2.88	2.48	4.7	29.4	33.2	28.5
R. M.	16-17	13.84	12.94	.716	4.52	3.85	5.2	32.5	32.6	27.8
R. M.	18-19	5.58	5.48	.296	1.86	1.62	5.3	33.1	33.3	28.9
J. D.	7-8	10.63	7.48	.619	4.41	1.70	5.8	36.2	41.3	15.9
J. D.	9-10	10.33	7.43	.603	4.07	1.69	5.8	36.2	39.3	16.3
J. D.	12-13	8.92	5.27	.625	3.67	1.28	7.0	43.7	41.1	14.3
J. D.	14-15	6.22	4.92	.347	2.21	1.30	5.6	35.0	35.6	20.8
J. D.	15-16	6.23	4.68	.399	2.16	.970	6.4	40.0	34.7	15.5
J. D.	17-18	10.98	6.33	.775	4.14	1.67	7.0	43.7	37.8	15.2
J. D.	18-19	6.67	4.12	.471	2.30	.920	7.1	44.4	34.5	13.8
	March, 1911									
J. H.	13-14	4.05	3.15	.225	1.042	.882	5.6	35.0	25.7	21.7
J. H.	14-15	4.55	3.80	.239	1.396	1.225	5.3	33.1	30.7	27.0
J. H.	17-18	3.20	2.50	.161	1.295	.670	5.0	31.2	40.4	20.9
J. H.	18-19	4.70	4.20	.217	1.380	.973	4.6	28.8	29.4	20.7
J. H.	19-20	4.20	3.50	.201	1.057	.844	4.8	30.0	25.2	20.1
J. H.	20-21	6.88	6.48	.282	2.340	1.853	4.1	25.6	34.0	27.9
J. H.	21-22	7.91	7.41	.291	3.632	1.818	3.7	23.1	46.0	23.0
J. H.	22-23	3.45	3.35	.132	1.343	.696	3.8	23.8	38.9	20.2

*Fat lump only separated, few small hard curds left in the feces.

It remains to discuss the data regarding the practical significance of the symptom. It has been stated before that the curds represent part of the food that has escaped being digested. It naturally follows that the injury occasioned by it to the economy of the organism may be very perceptible when the proportion of the non-utilized fraction reaches a high value. The review of Table 2 clearly demonstrates that such may be the case only on very rare occasions. Thus it is seen that out of seventeen stools, in nine the curds contained less than 1 gm. and not more

than 1.5 gm. of dry material. Calculating their protein content at 65 per cent., the loss of protein occasioned by the curd formation in these nine instances ranged between 0.06 and 0.95 gm. in twenty-four hours. In four stools the total dry weight of the curds reached from 2.0 to 3.0 gm. This resulted in a protein loss varying between 1.35 and 2.0 gm. in twenty-four hours. In four other stools the dry weight of the curds was between 3.15 and 4.65 gm., which meant a loss of protein of from 2 to 3 gm. Calculating the twenty-four-hour intake at 20 gm., it becomes evident that only on very few occasions was the loss resulting from the curd formation considerable. Generally, however, the appearance of curds in stools in itself does not lead to a perceptible decline in the utilization of the food-stuffs.

Furthermore, this conclusion is corroborated by the metabolism experiments. It is seen from Table 5 that the largest mass of curds was formed in the fifth period, which coincided with the period of the largest nitrogen retention. Of the two periods, fourth and fifth, with about equal protein intake, the mass of the curds was greater when the carbohydrate intake was of a lower, and the fat intake of a higher value. Comparing further the calorific value of the diet of the fourth and fifth periods, in which the protein intake was unchanged, one notices that in the fourth period the food utilization was less perfect, and yet it led to the formation of a smaller mass of curds.

On the other hand, the stools were completely free from lumps in the second period. In that period the food intake was of a comparatively lower calorific value, and in proportion to other food-stuffs rich in carbohydrates and very poor in protein. The retention of nitrogen in that period was very insignificant. In other words, the state of nutrition was in a less perfect condition when the stools were free from curds.

CONCLUSIONS

1. The "hard" or "casein" curds represent remnants of food, principally of protein nature, that have escaped being digested.

2. The exact mechanism of their formation as yet cannot be ascertained and they should be regarded as a peculiarity appearing in course of imperfect conditions of digestion.

3. The curds are not pathognomonic of any definite pathologic condition.

4. The loss of food occasioned by their formation and the impairment of the general nutrition resulting from it is insignificant.

5. In attempting to correct the state of digestion one should be guided by the general rules of infant feeding, paying only secondary attention to the appearance or disappearance of curds from the stools.

TABLE 5.—DANIEL L. PROTEIN RETENTION*

Number	Date	Intake in gm.			Intake in Per Cent.			Intake in Calories. Total Number Calories	Output.		Daily Average Nitrogen				Average Daily Gain in Weight	Description of Feces
		Carbohydrate, gm.	Fat, gm.	Nitrogen, gm.	Carbohydrate, Per Cent.	Fat, Per Cent.	Protein, Per Cent.		Urine, gm.	Feces, gm.	Absorbed, gm.	Retained, gm.	Absorbed, Per Cent.	Retained, Per Cent.		
	Oct. 1910															
1.	15-25	55.34	16.1	2.109	5.0	1.4	1.2	453	1.496	0.456	1.651	0.155	78.3	9.4	— 3.9	Partly pasty, smooth, gray-yellow and brown-yellow, partly watery, granular, green-brown. Hard curds present on three days.
2.	25-28	73.85	14.5	1.759	6.0	1.2	1.0	495	1.377	0.321	1.438	0.061	81.8	4.2	+12.3	Mostly formed and brown; a little thinner and green-brown. No hard curds present.
	Nov.															
3.	8-15	95.0	27.4	3.618	7.4	2.2	2.0	776	1.651	0.946	2.672	0.981	84.5	32.1	+28.7	Partly formed, smooth, brown; partly thinner, granular, yellow. Hard curds present on four days.
4.	15-22	84.04	18.0	4.831	6.3	1.0	2.55	695	3.201	0.809	4.022	0.821	83.3	20.4	+15.1	Like the feces of preceding period, generally more watery. Hard curds present every day, often large curds.
	Dec.															
5.	6-10	56.05	20.5	4.765	5.0	1.8	2.1	608	2.709	1.005	3.760	1.051	77.9	28.3	— 7.6	Mostly loose-watery (first day only formed), granular, with mucus, green-yellow, many large hard curds, some present every day.

Composition of Food

No.

1. 35 per cent. milk, 5 per cent lime-water, 60 per cent. water, milk-sugar to 5 per cent.
2. 30 per cent. milk, 5 per cent. lime-water, 65 per cent. barley water, milk-sugar to 5 per cent., 12 c.c. malt-soup extract.
3. 55 per cent. milk, 5 per cent. lime-water, 40 per cent. water, milk-sugar to 5 per cent, 30 c.c. malt-soup extract.
4. Fat-free milk 20 per cent., skimmed milk 55 per cent., lime-water 5 per cent., water 20 per cent., milk-sugar to 5 per cent., 16 c.c. malt soup extract.
5. Fat-free milk and full milk, 5 per cent. lime-water and water to make milk fat 1.8 per cent., milk protein 2.1 per cent., milk-sugar to 5 per cent.

After the present communication was completed for publication there appeared a paper by Brennemann[7] on the etiology and nature of hard curds. On the basis of many observations the author was led to the conclusion that curds appear in stools only after feeding on raw milk, and that the curds disappear as soon as the diet is changed to boiled milk. Brennemann takes for granted that the appearance of curds is the result of a serious fault of digestion, which can be corrected by changing the diet from raw to boiled milk. The observations of Brennemann may prove of practical and theoretical importance, but his work fails to furnish new information as to the clinical significance of the appearance of the curds. The present observations may be of some value whether or not the views of Brennemann are corroborated.

Without any desire to contradict, or to disagree with the views expressed by this writer, attention may be called to the fact that the first patient under our observation was fed on boiled milk.

METHODS OF ANALYSIS

Total Nitrogen was determined by the Kjeldahl-Gunning method. Samples of food mixtures, feces, and curds were dried on a water bath and then to constant weight in an air bath.

Sugar estimation was made volumetrically by the method described by Ettore Selvatici.[8] The procedure was the following: The proteins of the milk were removed by coagulation, and the clear filtrate was added from a burette into a definite volume of a boiling Fehling's solution. A small amount of potassium ferrocyanid is added to Fehling's solution previous to the beginning of titration. The sugar solution is added until the Fehling solution loses its blue color.

Starch content in barley water, barley jelly, and bean gruel was determined by its inversion into glucose. This was accomplished by heating the solution with dilute sulphuric acid in an autoclave. The sugar was titrated by the Pavy method.

Fat estimation was made by extraction with ether in a Soxhlet apparatus. For the determination of the total fat, the substance to be extracted was carefully mixed with phosphoric acid. Fat exclusive of soap fat was estimated directly on the unmixed feces. In the food, fat was estimated by extracting with ether in a Soxhlet apparatus. In order to remove the moisture from the ether extracts, the residues of these extracts were redissolved in chloroform, filtered through paper moistened with chloroform, and dried first on a water bath and then in a desiccator.

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7. A Contribution to Our Knowledge of the Etiology and Nature of Hard Curds in Infants' Stools, AM. JOUR. DIS. CHILD., 1911, i, 341.

8. Selvatici, Ettore: Bul. de l'Assn. des Chimistes de sucrerie et de distillerie, 1910, xxvii, 1179.

Ash-Estimation.—The dry samples of food or excretions were incinerated and the residue extracted with hydrochloric acid. The insoluble part was further ignited with the use of a few drops of strong nitric acid. The acid filtrate was added to the residue and evaporated to dryness.

The total acidity and volatile acids in the feces were determined in the following way: A known proportion of the moist feces was made into an emulsion by shaking in distilled water with glass beads, and then made to definite volume. An aliquot part was titrated for total acidity with decinormal sodic hydrate, phenolphthalein being used as indicator. To determine volatile acids, another portion of the emulsion was made acid to congo paper with dilute phosphoric acid and distilled with steam into a measured quantity of decinormal sodic hydrate until the dropping distillate was no longer acid; the unneutralized sodic hydrate then being titrated with decinormal sulphuric acid and phenolphthalein used as indicator.

HISTORIES

Daniel L., 7 months old, was admitted to the hospital Oct. 5, 1910. There was a history of protracted diarrhea and no gain in weight. He weighed 10 pounds, 8 ounces and was poorly nourished. Metabolism experiments were carried on from October 15 through October 28, from November 8 through November 22, and from December 6 through December 10, and the stools were collected for analysis until December 19. During the first period the weight was variable, being about the same at the end as at the beginning, 4,722 gm. The temperature was also variable. During the second period the child was looking well and the weight increased rather steadily from 5,003 grams to 5,310 grams. In the last period the weight was again variable, showing a slight loss at the end of the metabolism experiment and a small increase during the following days in which the stools were collected. During this time the child had low, irregular fever due to a cervical adenitis, which continued for some time after the conclusion of the experiment, accompanied by loose stools and falling weight. Subsequently the food was changed to one with less fat and protein and high carbohydrate with marked improvement and gain in weight.

Richard M., 2½ months old, was admitted to the hospital Sept. 6, 1910. He had not been gaining weight, and for ten days had had diarrhea with fever and some vomiting. He was poorly nourished but not emaciated, weighing 9 pounds, 6 ounces. He was used as a control for the Daniel L. experiments, his stools being collected from October 16 through October 28, November 8 through November 22, and December 6 through December 19. During all this time he showed a generally rising weight-curve, from 4,527 gm. on October 16 to 5,063 gm. on December 19. After the first period the stools were very good, though they frequently contained small hard curds, rarely large or numerous ones. He was discharged December 25, gaining and in good condition.

John D., 2½ months old, was admitted to the hospital Sept. 27, 1910, with a history of diarrhea and loss in weight. He was emaciated and rachitic. His weight was 5 pounds, 11 ounces. He began to gain almost at once and his stools improved. Successive increases were made in his food as his weight became stationary, on October 11, October 22, November 8, November 16, and November 23. Between November 8 and November 16 a peri-rectal abscess formed and he had fever but still continued to gain in weight. December 3 large hard curds began to appear in his stools. From December 6 through December 19 the stools were collected for analysis. During this time his stools were generally good, though they contained many large hard curds, and his weight increased from

3,508 gm. December 6 to 3,584 gm. December 18. Double otitis media developed from December 5 and erysipelas of the scalp from about December 18, with more or less fever, and he began losing weight. He was well of the erysipelas by December 29, but one ear was still discharging at that time. The gain in weight began when he was placed on a milk formula with low fat and protein and relatively high sugar.

Joseph H., 9 months old, was admitted to the hospital Feb. 6, 1911, weight 8 pounds, 3½ ounces. There was a history of constipation and considerable colic, and recently, vomiting and loss of weight. He was rather poorly nourished. There was no vomiting after admission but his weight remained at a standstill until February 24, when his food was increased. He served as a metabolism control from March 14 through March 23. During the experiment he gained slightly in weight, from 3,955 gm. to 3,994 gm. and had good stools, which, however, invariably contained hard curds, often large and numerous ones. He continued to gain in weight and have good stools and was discharged April 23, weighing 4,583 gm.

THE CALCIUM METABOLISM IN INFANTILE TETANY, WITH REPORT OF A CASE*

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Before discussing the rôle of calcium in the etiology of tetany it is important to summarize the present status of our knowledge of calcium in the healthy organism.

To illustrate how minute is the amount of calcium involved in metabolic processes, one needs but consider that the soft tissues contain 0.01 per cent. of the calcium in the body, the remainder forming 7.7 per cent. of the skeleton; thus, a child weighing 10,000 gm. would only have 1 to 2 gm. in the soft parts.

The calcium content of the brain, according to Quest[1] is relatively high in early infancy, but diminishes as the child grows older, most rapidly in the first few months, then more slowly. In the fetus he found 0.168 per cent. calcium, in a new-born child 0.107 per cent., in a 4 months child 0.072 per cent., at 16 months 0.074 per cent., and at 2½ years there was 0.067 per cent. The nitrogen content was fairly constant, being 10 to 12 per cent. of the dried powder. In the brains of three infants dying with tetany 10, 11, and 12 months old, however, Quest found the calcium content distinctly diminished (0.041 per cent., 0.047 per cent., 0.0535 per cent.). Aschenheim[2] found the percentage diminished in two patients aged 7 and 10 months, and approximately normal in a third case (0.0373 per cent., 0.0234 per cent., 0.0557 per cent.). Leopold and von Reuss,[3] however, did not find any change from the normal in two infants 4 and 5 months old, respectively (0.062 and 0.038 per cent.). Michel Cohn[4] also found the calcium content of the brain in one case of tetany normal. In animals suffering from tetany parathyreopriva it has been demonstrated by MacCallum,[5] Pexa,[6] and Aschenheim[2] that the calcium of the brain is diminished. Cooke,[7] on the other hand, found a slight increased calcium content in the brains of dogs dying of tetany. However contradictory the results may appear,

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*From the Chemical Laboratory of Cornell University Medical College.

1. Quest: Jahrb. f. Kinderh., lxi, 114.
2. Aschenheim: Monatschr. f. Kinderh., vii. 374.
3. Leopold and von Reuss: Wien. klin. Wchnschr., 1908, No. 35.
4. Cohn, Michel: Deutsch. med. Wchnschr., 1907, No. 48.
5. MacCallum and Voegtlin: Jour. Exper. Med., 1909, p. 119.
6. Pexa: Arch. f. Kinderh., 1910, No. 54.
7. Cooke: Jour. Exper. Med., 1910, No. 12, p. 45.

they may be better interpreted in the researches of Loeb who showed that it is not the presence of one salt alone in the solution, but its interaction with other salts which influences nerve irritability. Either a diminution of the Na or an increase of the Ca diminishes the irritability. This is well illustrated by Aschenheim in his third tetany brain, in which, although the calcium content is high, the sodium also being high causes the quotient of the fraction $\frac{Ca}{Na}$ to equal 46, whereas in normal brains it is only 19 to 26.

The application of calcium to the cerebral cortex has been shown by Sabbatani[8] to diminish its electrical excitability. Quest[9] was able by means of calcium-poor food to diminish the electrical excitability of the peripheral nerves in dogs, presumably by diminishing the calcium content of the nervous system. Stoeltzner[10] in an attempt to repeat this work, obtained contradictory results; and Pexa,[6] who fed puppies on a calcium-free diet, was unable to show any hyperexcitability of the peripheral nerves, although at autopsy the brains contained but one-fourth the normal amount of calcium. Rosenstern[11] attempted to attack this problem from the clinical side, and in cases of spasmophilic diathesis gave large doses of calcium chlorid (3 gm.) by means of which he was enabled to diminish the electrical excitability, the kathodal opening contraction rising within an hour to its maximum, the effect disappearing within twenty-four hours. Of twenty cases tested fifteen responded to the calcium and five were unaffected. To prove that it was the calcium and not the chlorid that was active, he used various other chlorid salts, but with them was unable to obtain results. Large doses of NaCl, on the other hand, increased the irritability. These influences on the electrical reactions have been investigated in the nerve-muscle preparation of the frog by Reiss[12] who showed that variations, i. e., a diminution in the calcium concentration of the solutions applied, could explain most of the electrical reactions of tetany with the exception of anodal closure.

An examination of the calcium content of the blood has also been carried out in order further to clear up the relationship of this substance to tetany. In parathyroidectomized dogs MacCallum and Voegtlin[5] found the amount diminished. Neurath,[13] using Wright's method, found that the calcium content of healthy children's blood was highest in early age and diminished as the child grew older, breast-fed children showing

8. Sabbatani: Quoted by Quest (see Note 9).
9. Quest: Wien. klin. Wchnschr., 1906, No. 27.
10. Stoeltzner: Neurol. Zentralbl., 1908, No. 2 (quoted by Escherich).
11. Rosenstern: Jahrb. f. Kinderh., ii, No. 2, p. 72.
12. Reiss: Ztschr. f. Kinderh., iii, No. 1.
13. Neurath: Ztschr. f. Kinderh., 1910, i.

a higher percentage than the artificially fed. Adding calcium to the food does not seem to increase the calcium in the blood. Parathyroidectomized animals also showed diminution of the blood-calcium. However, he emphasizes the fact that by Wright's method only ionic, i. e., precipitable calcium is estimated.

From the clinical standpoint this problem has been attacked by watching the therapeutic effect of doses of calcium in tetany. In post-operative tetany both in animals and man, large doses of calcium have been successfully used to relieve the symptoms. In the tetany of adults Curschmann and many others have had good results. In the infantile form Gerstenberger[14] got no results by the oral administration of calcium lactate, but his doses were only 2 gr. three times a day. Rosenstern[11] used it effectively by giving large doses. Intravenously calcium has yielded even better results.

At first sight it would seem that the most rational method of solving the whole problem would be by means of metabolism experiments; that is to say, ascertaining the total calcium intake and output. Although of undoubted value, this method has the disadvantage that it does not give any information as to the intermediate metabolism of the calcium, or as to how much calcium there is at any time available for the various parts of the body. It is surprising how little is known about the calcium metabolism, either in normal or in abnormal cases, and especially in cases of tetany.

The metabolism of normal breast babies has been better investigated on account of the ease with which it is possible to obtain a normal breast-fed child. Michel,[15] in a number of infants from 5 to 11 days old, found an absolute retention of calcium of about 0.2 mg., about 60 to 80 per cent. of the intake. In older children, aged 3 to 3½ months, Michel and Perret[16] and Schabad[17] have found slightly less retention, 150 to 200 mg., 40 to 50 per cent. of the intake. Langstein and Meyer[18] give as an average of various observations a retention in breast-fed babies of 176 mg. or 62.44 per cent. of the intake.

As far as bottle-fed babies are concerned, the figures vary from 1 to 5 per cent. retention (Cronheim and Muller[19]) to 44 per cent. Blauberg.[20] It must be added, however, that as Schabad remarks, the figures of Cronheim and Muller's cases are so low that they do not cover the calcium requirement of the organism. Langstein and Meyer give

14. Gerstenberger: Jour. Exper. Med., Mar. 1, 1911.
15. Michel: L'Obstetrique, Mar. 15, 1896 (quoted by Schabad).
16. Michel and Perret: Bull. Soc. d'Obst., March, 1899.
17. Schabad: Arch. f. Kinderh., lii, 1-3, p. 68.
18. Langstein and Meyer: Säuglingsernährung und Säuglingsstoffwechsel.
19. Cronheim and Muller: Biochem. Ztschr., ix, 79.
20. Blauberg: Ztschr. f. Biol., xl, 19.

the average retention as 44.4 per cent. These figures must be regarded as more or less arbitrary, as not enough cases have been examined nor have the durations of the various experiments been of sufficient length.

CASE REPORT

In a case of tetany which we had the opportunity of observing, the calcium metabolism was studied in three periods of three days each. The history of the case is as follows:

History.—Female child 1 year old, non-instrumental delivery, breast-fed till the age of 10 months; apparently normal until 9 months of age (could stand and made attempts to walk alone), when it suddenly began to have convulsions, which were repeated. For this it was taken to a hospital and remained there two months. Two weeks after it left the hospital it came under our observation. Its condition was as follows:

Physical Examination.—A poorly developed anemic female child with very moderate rachitis, extremities held in typical tetany position, with marked spasticity. Eyes staring, face mask-like; Chvostek extremely marked; Trousseau present. Galvanic irritability showed kathodal opening contraction at 1.0 milliampere. Physical examination of the lungs, heart and other viscera was negative; temperature 102, pulse 120, respiration 30; stools dyspeptic; urine showed a faint trace of albumin.

On admission the child was put in a metabolism bed, and a four-day experiment carried out, the first day being disregarded. It was given in three days 2,300 c.c. of milk, containing by absolute analysis 2.5 per cent. fat, 2.25 per cent. protein and 6 per cent. sugar. During this time and for several weeks afterward the child ran a temperature up to 102. Physical examination including ears, urine, etc., failed to show the cause for this. The Pirquet test was negative.

During the following month the child's food consisted of little milk and plenty of cereal. During this period, although the child took its food badly and lost in weight, the symptoms of manifest tetany improved considerably, so that the child lost its fixed staring expression, Trousseau could no longer be obtained, the Chvostek became less marked and kathodal opening contraction had increased to 3 milliamperes.

May 25 the child was again put in the metabolism bed. It was fed on a cow's milk mixture (fat 3 per cent., protein 2.2 per cent., sugar 6 per cent), of which it took 1,870 c.c. Its weight at this time was 12 pounds, 14 ounces. After four days the child was removed from the metabolism bed and put on a milk-free diet of soup and cereal for one week. Then it was given cod-liver oil 3i t. i. d., 10 ounces of milk a day, cereals, soup and bread and butter. The milk was then gradually increased. Under this regimen the symptoms rapidly improved, the temperature became normal, and remained so; kathodal opening contraction rising to 6 milliamperes, the only evidence of tetany being a slight Chvostek.

July 21 the third metabolism experiment was begun. During this the child received 2,250 c.c. milk, 120 gm. bread, 150 gm. rice, containing in all fat and protein as seen in the table. The child's weight at this time was again 14 pounds.

During the entire period of observation the stools were one to two a day, unformed, greenish-yellow, dyspeptic. The urine during the first few days of the illness contained a faint trace of albumin and a few hyaline casts. Subsequently the urine was normal.

SUMMARY

A child observed three months.

Experiment 1.—Manifest tetany; kathodal opening contracture 1 milliampere; fever; feeding raw cow's milk, calorically slightly insufficient.

Experiment 2.—Tetany markedly improved, kathodal opening contracture 3: fever: feeding raw cow's milk calorically insufficient through the child refusing nourishment.

Experiment 3.—After milk-free diet and cod-liver oil only symptom of tetany a slight Chvostek: kathodal opening contraction 6 milliamperes.

From Table 1 of the balances in these three experiments, it will be seen that although the child was fed a calorically insufficient mixture, the nitrogen balances were positive, 50 per cent. in the first experiment, and 38 per cent. in the second.

TABLE 1.—FAT AND NITROGEN IN AN INFANT WITH TETANY

	—Fatty Acids, K. and S.—				——— Total Nitrogen ———					
			Resorption,				**Retention,**		Resorption,	
Experiment	Intake, gm.	Feces, gm.	gm.	Per cent.	Intake, gm.	Urine and Feces, gm.	Per cent.	gm.	gm.	Per cent.
1 ...	50.46	5.72	44.7	88.7	8.13	4.06	4.07	50.0	7.71	88.2
2 ...	44.8	1.46	43.34	96.7	6.73	4.12	2.61	38.7	5.25	78.0
3 ...	93.6	4.8	88.8	94.8	14.32	7.14	7.185	50.0	13.64	95.2

The fat balance obtained by the method of Kumagawa and Suto, showed an intake of 54 gm. in Experiment 1, with resorption of 88 per cent.; the resorption in the second being much better (96 per cent.); in the third about 95 per cent., showing an improvement in the fat resorption in the last two experiments.

TABLE 2.—CALCIUM OXID BALANCE

		——— Food ———			— Urine —		— Feces —		Resorption		Retention	
Experiment	Duration, Days.	Amt., c.c.	Composition	Intake, CaO, gm.	Amount c.c.	CaO, gm.	Amt., gm.	CaO, gm.	Absolute, gm.	Per cent.	Absolute, gm.	Per cent.
1	3	2,300	2.5 F. 2.2 P. 6 S	2.921	635	0.0314	20.0	1.7834	1.376	38.9	1.1062	37.8
2	3	1,870	3 F. 2.2 P. 6 S	2.917	295	0.0288	7.0	0.8044	2.1128	72.4	2.084	71.3
3	3		Milk, Rice, Bread	3.918	1,026	0.220	16.0	1.426	2.492	63.6	2.272	58.7

The calcium metabolism as seen from Table 2 shows that in Experiment 1, with an intake of 2.9 gm. of calcium, there was an absolute resorption of 1.376 gm., or 38 per cent., and an absolute retention of 1.1062 gm. or 37.8 per cent. In the second experiment with practically the same intake there was only 0.8 gm. excreted in the feces, an absolute resorption of over 2.0 gm. or 76 per cent. and a retention of 71 per cent. The amount of calcium excreted in the urine in both of these experiments was about the same. In the third experiment, in which the intake was increased by 1 gm. of calcium, the amount of dried feces being almost the same as in Experiment 1 (16 gm. as compared with 20 gm.), the actual excretion was less than in Experiment 1, the absolute resorption

was 2.4 gm. as compared with 1.3 gm. in Experiment 1, and 2.1 gm. in Experiment 2, the percentage being 63 per cent., almost as large as in Experiment 2 and almost twice as large as in Experiment 1. The retention was 58 per cent. The amount of calcium oxalate in the urine of Experiment 3 is high, due to the unfortunate admixture of some of the feces. However, even if we assume that all the calcium found in this urine in Experiment 3 was derived from this contamination, nevertheless, by adding this to the calcium of the feces we still find the excretion less than in Experiment 1, although the child was getting 1 gm. more calcium by mouth.

It has been shown that the calcium output may be increased by fever,[21] and it might be argued that the comparatively low retention (37 per cent.) in our first experiment was due to this cause; however, during the second experiment the child ran a similar temperature and the retention had almost doubled.

Concerning the rickets, Schabad[22] has shown that as the rickets improves the calcium retention is bettered. In our case it seems hardly probable that the rickets was much of a factor, for the child had no enlargement of the epiphyses, had been able to stand, and had made attempts at walking at 9 months; in fact, the rosary was the only symptom. In a period of five weeks, during which time there had been no especial therapy or diet instituted for rickets (cod-liver oil was given only after the second experiment) and the child having lost weight, the improvement in the calcium retention could scarcely be attributed to improvement in the rickets.

The only cases comparable to ours are that of Cybulski[23] and those of Schabad.[24]

Cybulski's case was an infant 7 months old weighing 6,100 gm. (13.4 pounds). It was supposedly a non-rachitic infant suffering from manifest tetany. Cybulski did metabolism experiments of three periods of three, three, and four days each. The child received cow's milk in the first and woman's milk in the second and third experiments. From the figures in Table 3 it will be seen that there is a progressive improvement in the calcium retention, the figures showing first 20.8 per cent. retention, then 53.7 per cent., and last 87.2 per cent. The fat resorption, as in our experiment, also improved, but the nitrogen during the first experiment was negative.

The gradual increase in the amount of calcium retained might well be ascribed to the fact that the child received woman's milk, for this in itself ordinarily causes an increase in the calcium retention. Further-

21. Schkarin: Arch. f. Kinderh., 1905, xli.
22. Schabad: Arch. f. Kinderh., liv, Nos. 1-3.
23. Cybulski: Monatschr. f. Kinderh., November, 1906, p. 409.
24. Schabad: Monatschr. f. Kinderh., ix, No. 1.

more, the child during the first experiment was not in nitrogen equilibrium; moreover, it is very improbable that a child of the proletariat weighing only 13.4 pounds at 7 seven months should be free from rickets. In addition, Schabad calls attention to the absence of spastic extremities and the Trousseau sign, doubting as to whether the case is really one of tetany. It seems to us, however, that the electrical reactions undoubtedly classify the case as tetany, and it would seem more than likely that the convulsions could be ascribed to this cause.

TABLE 3.—CYBULSKI'S CASE. CALCIUM OXID BALANCE

Experiment No.	Duration, Days	Intake CaO, gm.	Urine CaO, gm.	Feces CaO, gm.	Resorption Absolute, gm.	Resorption Per cent.	Retention Absolute, gm.	Retention Per cent.
1.	3	1.756	0.0074	1.382	....	21.4	0.366	20.8
2.	3	0.997	0.0089	0.453	....	54.7	0.536	53.7
3.	4	1.531	0.031	0.1650	....	89.3	1.353	87.2

Schabad describes two cases of rickets complicated by tetany, 2½ and 3 years old, respectively, in which the calcium retention had sunk to 4 per cent. in the one and 15 per cent. in the other. By the administration of cod-liver oil and phosphorus he was able to raise the retention in the second case from 15 per cent. to 76.6 per cent., and coincidently noted the diminution in the electrical excitability — the kathodal opening contraction going from 2 to 6 milliamperes and the Chvostek and laryngospasm disappearing. It is difficult to compare these cases with ours, first, on account of the difference in ages, second, their marked rachitis and, third, the fact that Schabad's metabolism experiments did not include the stage of manifest tetany. It is seen that in his cases also the increase in the calcium retention and the improvement in the symptoms go hand in hand.

TABLE 4.—NITROGEN FRACTIONS IN URINE IN THE AUTHOR'S CASE OF TETANY

Date	Amt. c.c.	Sp. Gr.	Total Nitrogen, gm.	Urea, gm.	Urea N Per cent. Total N	NH_3 Nitrogen, gm.	NH_3N Per cent. Total N
April 18-19	130	1010	1.07	0.69	64.5	0.0585	5.47
April 19-20	205	1016	1.06	0.73	68.8	0.051	4.81
April 20-21	300	1010	1.08	0.84	77.8	0.072	6.66
May 25-26	95	1020	0.930	lost	....	0.04	4.3
May 26-27	65	1025	0.740	0.54	73.0	0.044	5.40
May 27-28	135	1012	0.970	0.607	62.6	0.091	9.39
July 19-20	380	1009	2.542	2.086	82.0	0.234	9.21

Haskins and Gerstenberger[25] in a child suffering from manifest tetany (negress 14 months old) found the calcium retention 11.7 per cent. Eight months later, after treatment, with the child apparently greatly improved, the calcium retention was 12.8 per cent.

25. Haskins and Gerstenberger: Jour. Exper. Med., March, 1911.

CONCLUSIONS

From the foregoing review of the subject, and analysis of our case, one might conclude as follows:

1. Sufficient data are not available to state definitely the absolute and percentual figures for calcium retention in the normal child.

2. The calcium in our case, as compared with the few normal cases on record, does not seem diminished.

3. From Cybulski's and our cases it appears that the calcium retention improves as the tetany disappears.

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REPORT OF BRAIN CASES

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During the past year a number of interesting cases involving the nervous system occurred in the service of my colleagues and myself at the Cook County Hospital, a few of which I herewith report.

CASE 1.—Johanna W., aged 8, of German parentage, but born in France. She was brought to the United States when 3 years old. No serious illness had occurred during the first few years of life. At the age of 6 she started to school, which she had to give up shortly thereafter on account of headaches. At that time the father noticed some change in the right eye and later a similar change in the left eye. The affection was of such a nature that she could see better in a dim light. She was treated with but little success for several months. The sight thereafter gradually improved and her headaches were relieved to the extent that she was again able to attend school. About a year ago the headaches returned and were very severe. Sleep was much disturbed and the only comfortable position when in bed was with the head lower than the trunk. She had not vomited before entering the hospital. Several months ago she had measles. This seemed to aggravate her condition. It was noticed about this time that she used her arms awkwardly, stumbled in walking, and had difficulty in talking. While out walking on one occasion she stumbled and fell. The injury did not seem severe, but immediately following the fall she had a slight convulsion which was not accompanied by unconsciousness. Her symptoms gradually grew worse. Walking became more difficult, her speech less distinct and she seemed to have some trouble in swallowing. She was brought to the County Hospital October 3, 1910, and walked into the examining room.

Family History.—Father and mother healthy. No history of syphilis. This was the only child.

Examination.—The patient was a girl of average height for her age, but undernourished; no tenderness of head nor rigidity of neck; face rather expressionless, mouth open; throat negative; no cervical adenitis; chest symmetrical and well formed.

Lungs: Percussion negative, auscultation negative.

Heart: Area not increased, tones normal, no murmurs.

Abdomen: Liver and spleen not enlarged. Abdomen otherwise negative.

Extremities: No edema or eruption.

Nervous System: Mentally clear, but unable to speak distinctly. Later she was able to answer questions only by nods and shakes of the head.

Muscular System (motor power): No absolute paralysis, but paresis. The left abducens was paretic; slight horizontal nystagmus.

Face: She moved only the left side of the face and that but little; could not protrude tongue nor corrugate lips; opens mouth; palatal reflex present but patient could not be induced to raise palate voluntarily.

Arms: Marked spastic paresis of left arm which was held in extension; slight spastic paresis of right arm.

Legs: Held in strong extension; partial flexion was possible but was never maintained; marked spasticity; the left was much more involved than the right.

Trunk: When placed in sitting posture patient had difficulty in maintaining her position. The spine showed scoliosis which was easily corrected.

Reflexes: Arm-jerks active, knee-jerks exaggerated. Babinski present on both sides. Oppenheim present. Gordon not elicited. No ankle or patellar clonus obtained.

Sensation: Touch intact; pain, analgesia of left side; temperature, intact; muscle sense intact; coordinated movements not well performed; patient had to be fed, but the difficulty seemed to arise from stiffness and weakness primarily. Taste and hearing so far as tested were all right.

Examination of left eye showed a retinitis.

Spinal puncture was made several days after admission to hospital. Pressure not increased; fluid clear. Prolonged centrifuging revealed very few polynuclear cells and lymphocytes and no bacteria.

Pirquet skin reaction was made and found negative.

Wassermann examination of blood was negative. No leukocytosis. Urinalysis was negative.

Subsequent Course.—In the subsequent course of the disease all symptoms became more pronounced and the patient passed into a state of profound coma, which continued for a few weeks before death, which occurred Dec. 27, 1910. She had in this period, several times a day, a slight general convulsion, during which the whole body would become rigid and the face flushed. Nasal feeding was necessary for several weeks. Decubitus developed on dependent parts of the body. The pulse was accelerated during her stay in the hospital, and she had at times a rise in temperature. The respiratory rhythm was disturbed during the period of coma.

Diagnosis.—In view of the general symptoms of cerebral pressure, namely headaches, retinitis, later vomiting and increasing unconsciousness, and in view of the focal symptoms, especially the paralysis of the right facial and the marked spastic paresis of the left arm and leg, in contrast to the slighter paresis of the right arm and leg and left facial, also the dysarthria and dysphagia, the diagnosis of a brain tumor located in the pons was made. In investigating the etiology of the tumor, it was found that the blood was negative to the lues reaction, and that she gave a negative skin tuberculin reaction. While this latter point could not be decisive against tuberculosis, a negative reaction might be of greater value in a diagnostic sense than a positive reaction. Consequently it was thought that the tumor was a glioma, or might prove to be a solitary tubercle.

Necropsy.—The postmortem was made by Dr. Boughton, whose diagnosis was as follows: 1. Glioma of pons. 2. Decubitus on both trochanters, right ilium, sacrum and external maleolus. 3. Parenchymatous degeneration and slight passive congestion of the liver. 4. Fatty degeneration of the kidneys.

It will be noted from the history of this case that fully two years before death symptoms of brain tumor started. The process had evidently existed for some time before distinctive localizing symptoms developed themselves. The importance of recognizing brain tumors early is self evident. If we can satisfy ourselves that they are not luetic, and to this end I would not depend on a negative Wassermann but also resort to antiluetic treatment, there is only one course of treatment open, and that is operation, first for the symptomatic relief, and second, if possible, for the removal of the growth.

CASE 2.—Wanda M., aged 8½ years, admitted to the hospital Nov. 16, 1910; of German parentage but born in South Africa. Three weeks before admission the child began to feel somewhat indisposed, did not care to go to school, lost her appetite, complained of headache, was feverish and vomited twice or three

times. The mother insisted on the child continuing in school for about three days following her first complaints. The headache grew worse and she was taken from school, with all symptoms continuing and exaggerated. In the course of a few days she began to tremble in the legs and arms. This was mild in character until about five days before entering the hospital, when the child began to tremble all over. She complained of stiff neck and back and said she had pain everywhere, but especially in the head, shoulders and arms.

Previous History.—Had had measles and small-pox.

Family History.—Negative. There was one other child who was healthy.

Examination.—The patient was a fairly well nourished girl of 8 years; the skin was pale and her expression was anxious. She breathed with the mouth partially open. She seemed to be in great pain, was restless and irritable. Every few minutes the pain was so great that she went into a semi-clonic condition all over the body and then cried out loudly, a sharp rasping cry, then quieted a little; the same thing was repeated in a short time. Her mind was clear. There was no defect of speech.

Head: The head was not enlarged and there was no evidence of injury about the scalp. The eyes were wide open. The pupils were dilated but reacted to light. There was no discharge from the eyes or nose. There was sordes on the teeth.

Neck: A few postauricular glands were palpable on the left side. The neck was rigid. When the head was raised the shoulders and trunk came up also.

Thorax: There was good resonance over the lungs on both sides and the boundaries were normal and mobile. A few moist râles were heard posteriorly.

Heart: Area and sounds normal; no murmurs or adventitious sounds.

Pulse: Soft, easily compressible, and accelerated.

Abdomen: Liver and spleen not palpable, no abdominal tenderness.

Genitalia: Negative. No enlargement of glands except as above noted.

Extremities: The arms were held in semiflexed position all the time and were kept in chronic clonic motion. The fingers could be flexed and the arms extended, adducted, and abducted, but the child complained of considerable pain whenever they were moved. The back was held rigid. The lower extremities were held in semiflexion. A decided Kernig was present on both sides.

Reflexes: Eyes reacted to light and accommodation. Knee- and Achilles-jerks somewhat exaggerated. Reflexes of upper extremity present.

Blood examination: Reds, 4,900,000; whites, 11,000; hemoglobin, 95 per cent.; index, 0.9.

Differential: Small mononuclears, 13; large mononuclears, 7; polynuclears, 80.

Spinal fluid: Examined Nov. 17, 1910. Amount, 16 c.c., under pressure; looked like water; centrifuged nine hours; a few lymphocytes but no bacteria found.

Urine, negative.

Wassermann reaction, negative.

The temperature on entering the hospital was 100.4 F. rectal; pulse 120.

Following spinal puncture the pulse-rate dropped to 90, to 80, and the rectal temperature remained below 100 F. All symptoms subsided, and in a few days the patient was convalescent.

Diagnosis.—The severe headaches, the rigid neck and back and the presence of Kernig's sign were sufficient to suggest meningeal involvement. She had a general tremor involving the head, trunk and extremities. The tremor was constant. It was greatly increased in voluntary movement or in touching the patient, as she was hyperesthetic. The tremor was coarse, the excursions being large. During movement of the patient the term semi-clonic would better describe it than tremor.

In regard to the lumbar fluid, the stained specimens made after centrifuging the fluid nine hours failed to reveal any organisms, and only a few lymphocytes. This might have been interpreted as a negative finding, or one suggesting a

tuberculous or luetic infection. A Wassermann reaction of the patient's blood was negative; consequently lues was strongly improbable.

The patient recovered rapidly following the lumbar puncture, so that tuberculosis could not be considered. There was no infection of the nose, throat, ears or sinuses that could be made out and to which the meningeal symptoms might have been considered secondary. She had what looked like a small burn over the left elbow, but there was no evidence of any considerable infection of it. There was no relation to be made out between this and the meningeal involvement. There had been no injury to the head nor was any intoxicant or narcotic to be considered in the matter.

After being in the hospital several days an examination of a vaginal smear showed it positive for gonococci. She was, therefore, transferred to the children's annex.

While we might speculate on the character of the meningeal involvement, we certainly had no definite point on which to make an etiologic diagnosis, directly or indirectly. It might be consistent for us to consider the possibility of serous meningitis, because she presented some of the symptoms of meningitis, because the lumbar fluid was under pressure, because it was practically negative on examination, and because she recovered promptly following puncture. It possibly had some toxic or infectious origin, a view which might be reinforced by the existence of a slight temperature, but we had no leading points on which to base it nor with which to associate it.

CASE 3.—M. L., girl aged 13, Jewish, born in Russia.

Family History.—This was the oldest and only living child. The mother's second pregnancy terminated in a miscarriage at the third month. The third child born was healthy at birth but died at six months of summer complaint. The result of the fourth pregnancy was a dead-born child. No insanity on the father's or mother's side so far as known. Parents were both in good health.

Personal History.—Her early development in regard to walking and talking proceeded about the same as most children. She was bright and as quiet as the average child. The patient had brain fever when 2½ years old and was sick for three months. At six years she had scarlet fever, but recovered entirely. She never seemed to care much for the company of children, preferring to be with older people. She started to school at 8 years but did not learn well.

Last summer (1910) the patient was attacked by four children, who threw her down and beat her. Her cries brought a neighbor, who found her greatly frightened, crying and trembling all over. When the mother returned home in the evening she was still greatly agitated. That night she slept but was very restless. On the following day, Tuesday, she went with other children for a boat ride. On returning home the mother thought she appeared sad. She answered questions and talked voluntarily. Wednesday evening, when getting out of the bath tub, she fell and struck her head. The mother noticed on helping her up that she was very pale, but she complained but little of her head hurting. When the mother returned from her work Thursday she found the patient crying. She said she did not want to live, because the children annoyed her. That night she slept but little, cried a great deal and talked in this same strain.

On the following day she stopped talking and maintained silence for ten days. She would not leave the house, would seldom answer a question, and if so, with a grunt, and would not eat unless her mother insisted on it. At the end of ten days she suddenly became very talkative and noisy; she talked in a rambling

way and would repeat almost everything that had been said to her during her quiet spell. She wanted to go to parks, nickel shows, etc., and wanted to become an actress.

Then periods of complete silence and agitation alternated regularly every ten days, for six months. She was taken to a sanatorium, from which she escaped after four weeks. She did not appear to improve while there, but after returning home there was a period of three months in which she seemed to do much better, did not have her mute spells and was quieter in general. While in the sanatorium it was noticed that the spells of silence and agitation were not so uniform. At no time had she had delusions or hallucinations.

Following the three months improvement, the mother noticed one day while the child was at the piano that a change came over her. She stopped playing and looked strange. On the mother inquiring the cause of the change she said her head hurt. After awakening on the following morning she did not speak for thirteen days. During this time she started to menstruate for the first time. Following a similar alternating period of agitation, she became quiet and during this time she also menstruated. She was subsequently removed to the Elgin Asylum.

Examination.—Lungs, heart and abdomen negative; urine negative.

Nervous System: Head did not show any evidence of past injury. Eyes reacted to light and accommodation.

Muscular System: No paresis or paralysis.

Sensation: No disturbance detectable.

Reflexes: Present but not exaggerated. No hysterical stigmata could be made out.

The family history was strongly suggestive of lues. The girl, however, did not present any evidence of luetic lesions past or present.

The mental change followed closely on the fright and head injury. The alternating periods of silence and agitation followed each other with great regularity. She showed a remarkable memory during her periods of agitation, not only of what was said during her spells of silence, but of early events in her life, of her native city, Odessa, etc. She was also physically restless at these times, while in her periods of silence she would stand in one position for hours.

In view of the clinical history of this case the question of diagnosis would probably consider circular insanity and dementia præcox. The alternating character of the periods of depression and emotional excitement would strongly suggest a circular form of insanity. However, such a picture might occur in the early stage of dementia præcox.

During her periods of depression, there seemed to be a complete suspension of mental activity, lack of interest in her surroundings and no tendency to physical effort, all of which points are strongly suggestive of dementia præcox. At no time, however, had she had the hallucinations or delusions that are frequently present in the beginning of dementia præcox.

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SPLENOMEGALY (GAUCHER)

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It is the object of this paper to report the history of a family, several members of which suffer from a type of splenic enlargement usually designated as that of Gaucher, and to make a critical study of all the reported cases of this disease, with the hope of proving that although these cases resemble other diseases, yet closer study gives overwhelming proof that they must be classed by themselves as an entirely distinct and separate disease. The diseases from which it is especially to be differentiated are splenic anemia in adults and von Jaksch's disease in infants. Under the term "splenic anemia," Osler includes all those cases of idiopathic enlargement of the spleen associated with anemia, which were formerly classified as splenic pseudoleukemia, anemia splenica, lymph adénie splénique and splenic form of Hodgkin's disease. He states: "I purposely have not spoken of anemia with enlarged spleen in very young children, a subject which requires separate consideration."

The entity of splenic anemia is not at all granted. Stengel in his paper on "Splenic Anemia" says: "I have elsewhere expressed doubt regarding the individuality of such a condition and the conviction that many different sorts of diseases have been classed under this term."

REPORT OF CASES

Family History.—Father and mother both living and well; there is no tuberculosis in the family; nor could any luetic history be obtained. The mother has given birth to four children, and has had no miscarriages nor stillbirths. Anna, the oldest child, is 11 years old. Lilly died in 1909 at the age of 8; Freda is 9 years old, and Max, the youngest, is 4 years old. Wassermann reaction of mother negative.

The boy came to my notice at the Vanderbilt Clinic June 10, 1911 (in the service of Dr. C. Herrman) where he was brought for treatment on account of a progressive enlargement of his abdomen. The mother stated that an older child (Lilly) had had a similar condition and that that child died two years previously; as this child (Lilly) had been at Mt. Sinai Hospital (1906) at a time when I was a member of the house staff of this hospital, I can recall the case very clearly. At the hospital no diagnosis was made. This child was also treated at the Vanderbilt Clinic in 1904, and notes of the case show that at that time the child had a much enlarged spleen and liver. No diagnosis was made at the time.

CASE 1.—Anna: Physical examination of this child revealed nothing abnormal; she was in good physical condition, the liver and spleen were not enlarged.

CASE 2.—Lilly: This child died two years previously at the age of 8; she had measles at 2 years; no other diseases of infancy; no history of malaria. At 4 years the child began to be troubled with enlargement of the abdomen; also complained of dragging sensation in the side and weakness. The abdomen was progres-

sively increasing in size; at the time the child was at the hospital the liver was 10 cm. below the free border of the ribs, and the spleen was beyond the median line and well filled the left iliac cavity. The child had at no time any hemorrhages nor oozing from the gums, and there was no discoloration of the face.

The blood examination showed the following:

Red blood-cells, 3,440,000; white blood-cells, 6,200; hemoglobin, 68 per cent.; differential count, normal; no abnormal cells present.

Mixed treatment, arsenic and *x*-ray, were ineffectual and the child was discharged from the hospital unimproved. A few months later the child suddenly developed dyspnea and cyanosis and succumbed before a physician could be called (Fig. 1).

CASE 3.—Freda: The third child was 9 years old; she had no diseases of infancy, and there was no history of malarial infection. During the last year she complained of pain in the abdomen. Physical examination showed the liver to be enlarged; the edge was palpable 2 inches below the free border of the ribs. The spleen was not enlarged and the edge was not palpable. The blood examination

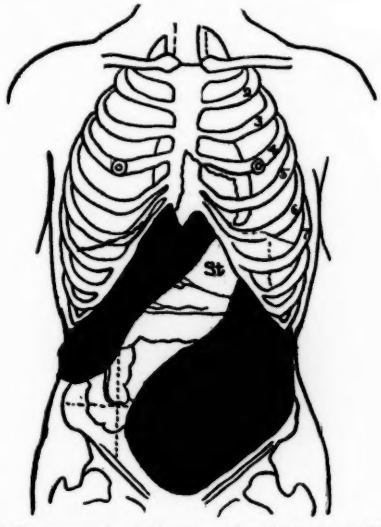

Fig. 1.—Enlargement of liver and spleen in Lilly, Case 2.

showed the following: Red blood-cells, 4,344,000; white blood-cells, 7,000; hemoglobin, 75 per cent.; differential count, normal; no abnormal cells present.

There was no pigmentation of the skin, and no history of any hemorrhages nor oozing from the gums or lips; there was no bone tenderness and the lymph-glands were not enlarged. Whether Freda was suffering from the same disease as Lilly and Max (the latter case to be described later) only the future will reveal; but she had a much enlarged liver which cannot be attributed to any other cause; whether she will develop a large spleen later cannot be definitely stated (Fig. 2).

CASE 4.—Max: The fourth child was a boy 4 years old; he had no diseases of infancy; he was not rachitic, and there was no history of malarial infection. About one year previously he was thrown down by a horse; at this time the attending physician told the mother that the child had a large liver and spleen; since that time the mother noted a progressive enlargement of the abdomen and brought the child to the clinic on that account. Examination of this child showed there was a distinct discoloration of the face; the color varied from light yellow to golden hue. The pigmentation was most marked at the bridge of the nose and around the eyes. There was no discoloration of the conjunctivæ. There was a

general languid appearance, and the child moved about very slowly; the child did not complain of any pain nor was any tenderness to be elicited in the chest or abdomen. Heart and lungs were normal. The liver was much enlarged and the edge was palpable at the umbilicus; there was no tenderness over the liver. The spleen reached to 2 inches below the umbilicus and extended over the median line to the right as far as the mammary line.

The blood examination showed the following: Red blood-cells, 2,208,000; white blood-cells, 5,000; hemoglobin, 35 per cent; differential count, normal; no abnormal cells found. There was no history of hemorrhages; no bone tenderness, and the lymph-glands were not enlarged (Fig. 3). Wassermann reaction negative.

It is my object now to epitomize all the cases reported in the literature and to try to make a critical study, with the hope of collecting data to show that they have much in common, and that they differ greatly from other similar diseases, and that, in fact, "Splenomegaly (Gaucher)" is an entity.

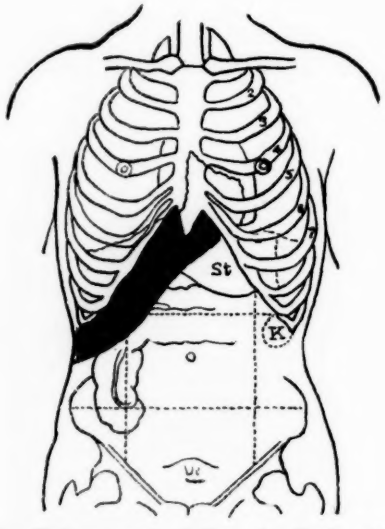

Fig. 2.—Enlargement of liver in Freda, Case 3.

GAUCHER'S CASE.[1]—Female, aged 32, died of intercurrent pulmonary tuberculosis; splenic enlargement since 7 years old, accompanied by enormous enlargement of liver; repeated epistaxis; ulceration and oozing of blood from the gums. The blood showed simple anemia. Necropsy showed tuberculosis of lungs and peritoneum, moderate enlargement of liver, large spleen (4,770 gm.); lymph-nodes normal.

PICON AND RAMOND'S CASE.[2]—Female, aged 32, with a history of menorrhagia, abdominal pain, swollen and bleeding gums; operated on for suspected fibroid uterus; large spleen found; spleen was removed; patient recovered from operation. The blood showed simple anemia; spleen weighed 2,800 gr. Four years previous to operation there was a history of trauma to abdomen.

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1. Gaucher: Thése de Doctorat-Splenomegalie Primitif; Epithelioma primitif. de la rate, 1882.

2. Picon and Ramond: Hypertrophie de la rate, Arch. de méd. exper. et Anat. Path., 1896.

COLLIER'S CASE 1.[3]—The spleen was taken from the body of a female child aged 6 years, who was admitted to Radcliffe Infirmary April 4, 1894, and died there Sept. 7, 1894. The mother stated that the child was healthy up to 8 months, when she had a severe illness the nature of which is not known. When 2 years old the abdomen was noticed to be enlarged, and was steadily increasing in size. The blood was examined, but no excess of white blood corpuscles was found. The red blood corpuscles were misshapen and did not form good rouleaux. Slight bending of ribs, but no other signs of rickets, was present. Death seemed to be accelerated by an attack of "epistaxis and sickness." The mother stated that she had seven children, four of whom died previously to this one—one of inflammation of the lungs, two of consumption of the bowel and one in the infirmary six years previously.

Necropsy showed no tuberculosis: spleen weighed 4 pounds 2 ounces; child 23 pounds; all mesenteric glands enlarged.

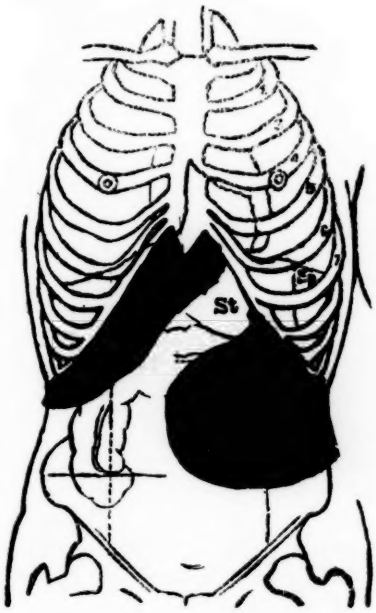

Fig. 3.—Enlargement of spleen and liver in Max, Case 4.

COLLIER'S CASE 2.—The sister died at an infirmary Nov. 27, 1888; was admitted with bronchitis and enlarged spleen; gradually lost strength and died of exhaustion. No tuberculosis was found on necropsy. The spleen was found to be enormously and uniformly enlarged: weight 25 ounces. On section it was found congested and its substance was firm though somewhat friable. (Reported Jan. 16, 1895.)

WEICHSELBAUM'S CASE.[4]—Male, aged 21, soldier; no history given; spleen removed by operation.

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3. Collier: A Case of Enlarged Spleen in a Child Aged 6, Tr. London Path. Soc., 1895.

4. Weichselbaum: Primar multiple Endothelsarkom der Milz, Virchows Arch. f. path. Anat., 1881.

BOVAIRD'S CASE 1.[5]—Female, aged 3; father and mother living and well; nine children; one stillbirth at eight months; four died during infancy from summer complaint; five are living; no previous diseases.

For about one year the mother noted enlargement of abdomen, steadily increasing; also noted a mass on right side of abdomen as well as on left; was sent to hospital; the child was of normal stature, well nourished, not anemic; skin dusky; some signs in the lungs; liver dulness from fourth space to 3 inches below free edge of ribs; edge was hard and sharp; spleen extended to within ½ inch of median line at umbilicus; lower end on a level with anterior superior spines of ilium; edge sharp and hard; nodes in each axilla; one node the size of a bean; both sets of inguinal glands were slightly enlarged, not tender, freely movable. Blood, 4,400,000 reds, 9,000 whites, hemoglobin 75 per cent.

TABLE GIVING ANALYSIS OF CASES

Author	Case No.	Sex	Age	First Splenic Enlargement Noted at Age of	Time Under Observation, Years,	Spleen Enlarged	Liver Enlarged	Lymph-Nodes	Red Blood Corpuscles	White Blood Corpuscles
Gaucher	1	F	32	7	25	Much	Much	Normal	Simple Anemia	
Picon and Ramond	2	F	32	?	?	Much	?	?	Simple Anemia	
Collier	3	F	6	2	4	Much	?	?	R.B.C misshapen No excess of W.B.C	
Collier	4	F	10	?	?	Much	?	?	?	?
Weichselbaum ...	5	M	21	?	?	Much	?	?	?	?
Bovaird	6	F	3	3	3	Much	Much	Enlarged	4,400,000	9,000
Bovaird	7	F	13	3	13	Much	Yes	Enlarged	2,880,000	4,000
Brill	8	M	9	?	?	Much	?	?	?	?
Brill	9*	M	31	15	19	Much	Much	Normal	3,200,000	8,240
Brill	10	F	34	19	19	Much	Much	Normal	4,800,000	7,168
Schlagenhaufen ...	11	F	41	5	36	Much	Slightly	Normal	4,700,000	1,300
Schlagenhaufen ...	12	F	33	20	13	Much	Slightly	Normal	5,735,000	3,435
Marchand	13	F	?	?	20	Much	Much	Normal	No leukocytosis	
Reuben	14	F	8	4	4	Much	Much	Normal	3,440,000	6,200
Reuben	15	F	9	—	1	None	Much	Normal	4,340,000	7,000
Reuben	16	M	4	4	1	Much	Much	Normal	2,208,000	5,000

*Ascites present. No jaundice in any case.

The child was in the hospital from Nov. 28, 1896, to Dec. 22, 1896; seemed quite well; chest signs cleared up; gained 1¾ pounds. Treatment, Fowler's solution.

Some time prior to July 26, 1899, the child had been failing; nose bled frequently and quite severely; lost flesh; face and hands had become bronzed. At this time—age 6 years—across the bridge of the nose and on the cheeks and hands there was diffuse bronzing of the skin; abdomen spare, prominent; superficial veins enlarged; liver extended from sixth rib to within 1½ inches of umbilical line. The spleen later extended into and filled the right iliac fossa. The glands, inguinal, cervical and axillary, were slightly enlarged. Blood, 4,180,000 reds, 14,000 whites, Hgb. 62 per cent. In December, 1899, the child had not improved but was able to attend school.

5. Bovaird: Primary Splenomegaly—Endothelial Hyperplasia of the Spleen—Two Cases in Children; Autopsy and Morphological Examination in One, Am. Jour. Med. Sc., 1900.

BOVAIRD'S CASE 2.—Female, sister of above patient, aged 13, had measles at 5 years; at 7 an eruption on the scalp. At the age of 3 the mother noticed an enlargement of the abdomen which progressively increased in size, and also pigmentation of the skin, especially across the nose. The child complained of a little discomfort from the enlarged abdomen and was a little short of breath. When admitted to the hospital she was small in stature for her years, fairly well nourished, skin dusky; across the nose, underneath the eyes and on the upper lip there was a decided brownish pigment. Liver dulness extended from the fifth space to the margin of the ribs; edge could not be felt; superficial veins were dilated. The spleen extended to 1 inch above the umbilicus, passed obliquely to the anterior superior spines and was hard and smooth. There was slight enlargement of right inguinal nodes, moderate enlargement of both submaxillary and one pal-

ASES OF SPLENOMEGALY (GAUCHER)

Pigmentation	Location of Pigmentation	Hemorrhages	Splenectomy	Autopsy	Still Living	Weight of Spleen, gm.
?	?	Epistaxis, bleeding gums	..	+	..	4,770
?	?	Menorrhagia, bleeding gums	+	..	..	2,800
?	?	?	..	+	..	2,070
?	?	?	..	+	..	880
?	?	?	+	..	..	....
Bronzed	Fase, nose, hands	Epistaxis	..	..	+	....
Brownish	Nose, face	?	+	+	..	6,200
?	?	?	..	..	..	....
Light Yellow	Face, hand, neck	Epistaxis	..	+	..	5,280
Brown-Yellow	Face, hand	Epistaxis, oozing gums	..	+	..	7,000
Brown	Face, lower extremity	Gums epistaxis	..	+	..	3,510
Bronzed	Cheeks, forehead	Epistaxis	..	..	+	....
Icteric-Brownish	?	?	..	+	..	2,720
Roman Gold	Face	—	..	..	..	....
.....	.....	—	..	..	+	....
Roman Gold	Face, hands	Epistaxis	..	..	+	....

pable node in each axilla; no enlargement of the epitrochlear nodes. Blood, 2,880,000 reds, 4,000 whites, Hgb. 60 per cent. Mixed treatment was tried without effect. Three years later the condition was the same; the child did not grow in stature nor gain weight. Readmitted to the hospital in 1899, aged 16 years. The patient was fairly well nourished, face and hands deeply bronzed; liver dulness began at the sixth rib; edge 1½ inches below the free costal margin; the spleen filled the whole of the left iliac fossa. Blood, 3,550,000 reds, 7,000 whites, Hgb. ? Splenectomy was performed May 17, 1899, by Dr. A. J. McCosh. The patient died three hours after operation. The spleen weighed 12½ pounds.

BRILL'S CASES.[6]—Father and mother both well; had six children; eldest in perfect health; second died at 3 years of marasmus and diarrhea; third, one of the cases here reported; fourth in good health; fifth, second case here reported; sixth died at the age of 9 years.

6. Brill: Primary Splenomegaly, with a Report of Three Cases Occurring in One Family, Am. Jour. Med. Sc., 1901, 1905, 1909.

CASE 1.—Male, aged 9 years, died with enlargement of the spleen.

CASE 2.—Male, aged 31 years; no malaria, rheumatism nor tuberculosis; had persistent patch of erythematous papules which extended from the malar prominence of one side across the skin covering the nasal bone to the same prominence on the other side, first observed in 1885; no splenic enlargement till 1889; occasional epistaxis. In 1899 first enlargement of liver noted; also a peculiar hard, ochre-colored, wedge-shaped infiltration of the sclerotic of the nasal side of each eye; lost weight slowly but progressively. In 1899-1900 the skin of neck, face, and hands began to be dusky (light yellow to brownish tint). In 1893 blood examination showed normal red and white count; Hgb. up to 1900 never lower than 80 per cent. In 1900 the progress of disease became rapid. The patient had an acute attack of colitis. After convalescence hemorrhagic furuncles appeared.

In 1902, pain appeared in the lower end of the left tibia; in June, 1902, the patient had a malarial attack; epistaxis became frequent. Blood, 3,200,000 reds, 8,240 whites. Hgb. 45 per cent. There was no increase in size of spleen during the attack of malaria.

In 1903 pain appeared in the ankle. Blood, 4,400,000 reds, 5,240 whites, Hgb. 55 per cent. The spleen extended 3 cm. beyond middle line and filled the iliac cavity. In 1904, ecchymoses appeared on the fibula. In March, 1904, there were dyspnea, fever and pericarditis. The patient died March 30, 1904, aged 34 years. Ascites was present for forty-eight hours before death; the glands were never enlarged. The spleen weighed 5,280 gm. Discoloration of the skin was limited to face, neck, wrists, and hands. There was no bone tenderness.

CASE 3.—Female, aged 34. Observed since 1885. In 1888 the spleen was enlarged; liver not enlarged; no glands. In 1895 the blood count was 4,800,000 reds, 7,168 whites, 80 per cent. Hgb. In 1896 the patient had typhoid fever. The color of the skin was brownish-yellow. There was frequent oozing from the gums; increasing anemia, increasing emaciation and increasing enlargement of the abdomen; infrequent attacks of epistaxis and oozing from the gums. Blood, 3,500,000 reds, 3,600 to 5,200 whites, Hgb. 35 per cent.

The patient complained of dyspnea. Sept. 12, 1904, the patient was thrown down and sustained a laceration of the brain and fracture of the skull; died same night. The spleen weighed 7,000 gm.

SCHLAGENHAUFEN'S CASE 1.[7]—Female, aged 41. The mother of the patient stated that the patient had enlargement of the spleen for a long time. At the age of 5 she was sent to the hospital on that account; at 21 brown pigmentation of skin of face was noted. Frequent bleeding from the gums and nose occurred. In January, 1906, the patient looked emaciated, weighed 39 kilos and a diffuse brown pigmentation of face and lower extremities was present. The spleen was enormously enlarged; liver slightly enlarged; sternum tender on pressure.

Blood: Jan. 2, 1906, 4,700,000 reds, 1,300 whites, 65 per cent. Hgb.; Feb. 6, 1906, 4,024,000 reds, 2,200 whites, 58 per cent. Hgb.; March 3, 1906, 800 whites.

May 20 the patient came in a dying condition to the hospital and died the same night. May 21, 1906, necropsy showed tuberculous glands of the neck with fistula; the spleen weighed 3,510 gm.

SCHLAGENHAUFEN'S CASE 2.—Female, aged 33, sister of the above patient, at 20 suffered from dysentery. At this time she was told by her physician that she had a large spleen. Since her tenth year her face was bronzed; had frequent nosebleeds; on cheeks and forehead broad, and under eyes, small patches of brown pigmentation; spleen enlarged to crest of ilium; liver somewhat enlarged; nodes palpable; blood, 5,735,000 reds, 3,435 whites, Hgb. 88 to 90 per cent. The patient felt fairly well and was able to work.

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7. Schlagenhaufen: Verhandl. d. deutsch. path. Gesellsch., 1906.

MARCHAND'S CASE.—Female, with a splenic tumor for twenty years; liver also enlarged; tendency to hemorrhage was noted; skin icteric; brownish color; no leukocytosis. The spleen weighed 2,720 gm.

ETIOLOGY

Family Incidence: The disease is undoubtedly congenital and usually affects more than one member in the same family. Of the cases here collected, there are three members affected in two families; two members affected in three families.

Sex: Many more females are affected than males. Of the sixteen cases here reported, twelve are females, four males.

Age: The youngest reported was 3 years, the oldest 41 years. Five of the patients were between 30 and 35 years; seven were between 3 and 10 years; one between 10 and 20 years; one between 20 and 30 years; one, age not given.

PATHOLOGY

In eight of the sixteen cases autopsies were performed, and in three the spleens were examined after removal by operation. In the eight autopsies performed the findings in the spleen and liver were almost identical in all the cases. Bovaird was first to note similar changes in the mesenteric glands; and Mandelbaum and Libman were the first to find the endothelial hyperplasia in the bronchial and retroperitoneal glands, and in the bone-marrow.

Macroscopic Examination.—The liver is firm and smooth over the greater part, and is usually much enlarged; there is evidence of old and new infarcts; on section it is chocolate colored and there are numerous small white areas.

The spleen is much enlarged; the form of a normal spleen is retained; on the external surface fibrous adhesions are present; the surface is white to yellow, and in general is smooth; the fibrous coat is thickened; the substance is firm and resistant.

The appearance of the cut section varies; a central section shows firm yellow-white areas of pyramidal form; the remainder has the appearance of normal spleen.

Lymph-nodes are enlarged and soft; on cross-section the central parts are deep red and the periphery is pale.

Microscopic Examination.—Spleen: Sections from hard white areas differ from other sections that have the appearance of normal splenic tissue. In the latter sections there are large irregular spaces filled with large, brightly stained cells. The walls of these spaces consist in part of a delicate line of connective tissue with infrequent small oval nuclei; adjoin-

ing spaces communicate with one another by narrow passages. The cells within the spaces are very large, and are of varied shape; many lie free in the spaces; these cells are round or oval. The nuclei vary considerably in size and shape; as a rule, they are very small in proportion to the size of the cell. There are also in some places giant cells. These spaces are the pulp spaces of the spleen, and the cells that fill them have sprung from their walls. The normal pulp cells have disappeared. On the other hand, the Malpighian bodies are met with almost unchanged. The connective tissue in some places is so abundant that the spaces contain but one cell.

Sections from the white areas consist of dense connective tissue. In part this is infiltrated with small round cells; in other parts seems to be made up of a meshwork of capillaries.

Capsule and trabeculæ are greatly thickened. The blood-vessels are surrounded by a greatly increased quantity of connective tissue.

The Malpighian bodies "seem very little affected by the great changes that have gone on about them."

Liver: The capsule is thickened and the connective tissue throughout the organ is increased; the capillaries contain considerable pigment; the liver cells are normal; the bile ducts are normal; in the blood that fills the branches of the portal vein there are large cells of the type seen in the pulp spaces of the spleen. Endothelial cells are mostly seen in the interlobular connective tissue spaces; a few are seen in the lobule proper.

The Lymph-Nodes: These are swollen, red and soft; each follicle is outlined by a wall of brightly shining, dark pigment. The part of the lymph-sinus not occupied by pigment is nearly completely filled with the above-described endothelial cells.

Bone-Marrow: Cells identical with those found in the above-described organs are also found in abundance in the bone-marrow.

SYMPTOMATOLOGY

1. Enlargement of the spleen. One of the first symptoms and signs noted by the parents and the patients is a mass in the left hypochondrium; this mass is not tender. There is a gradual but progressive enlargement of the spleen. The increase in size of the spleen in this disease is surpassed only in splenomyelogenous leukemia. It is surprising how large the spleen may become without causing any great inconvenience to the individual; and the enlargement of the spleen takes place a long time before any secondary symptoms arise. In many cases the condition is discovered accidentally, when the patients apply for treatment for other ailments. In almost all the cases the spleen passes the median line and the lower portion well fills the iliac cavity. The smallest spleen weighed 880 gm. The largest spleen weighed 7,000 gm. The average of nine

spleens was 4,000 gm. (see table). The earliest age at which the first splenic enlargement was noted was 2 years. The oldest 20. In two the splenic enlargement was noted at 3 years; in two at 4 years; in one at 5 years; in one at 7 years; in one at 15 years; in one at 19 years; in one at 20 years.

2. Enlargement of the liver. In all the cases there was a concomitant enlargement of the liver. In some cases the liver increased in size proportionally to the spleen; in others it was slightly enlarged. But in all the cases there was some enlargement of the liver. In all but one of the cases the hepatic enlargement was secondary to the splenic enlargement; and in all but one of the cases the spleen was larger than the liver. In Case 15 (see table) the liver is more enlarged than the spleen.

3. Enlargement of the lymph-nodes. The lymph-nodes are usually normal. In Cases 2 and 3 (see table) there was slight enlargement, but I think we cannot positively rule out specific infection. There is a history of a stillbirth at eight months and four deaths in early infancy in that family. I am of the opinion that the glandular enlargement was probably due to an underlying syphilitic infection. Patient 11 had tuberculous glands of the neck.

4. Blood changes. The blood shows usually a mild anemia; the anemia varies in severity from time to time, and is subject to various grades of improvement; only in later stages of the disease does it become progressive.

The lowest red blood-cell count was 2,880,000; the highest, 5,735,000; the average of seven cases, 4,100,000.

The lowest white blood-cell count was 1,300; the highest, 9,000; the average of seven cases, 5,600.

The lowest hemoglobin estimation was 45 per cent.; the highest, 88 per cent.; average of seven cases, 70 per cent.

Differential counts of six cases showed nothing abnormal.

Bone tenderness was noted in three of the sixteen cases.

5. Abdominal pain, fulness and a dragging sensation was noted in eight cases.

6. Pigmentation is described, varying from a brownish, bronzing and Roman gold color to a yellow, light-yellow, icteric hue. The pigmentation is seldom diffuse and usually affects the face, neck and extremities, the bridge of the nose, around the eyes and the cheeks being favorite spots.

7. Hemorrhages, as a rule, are not severe, but usually in the form of epistaxis or oozing from the gums; usually frequent. Patient 2 had menorrhagia. Patient 9 had hemorrhagic pericarditis, of which he died.

8. Jaundice and ascites are usually absent. Jaundice was present in two cases; ascites in Patient 9 for forty-eight hours before death.

COURSE OF DISEASE

The course of the disease is slowly progressive, with a certain number of remissions. The shortest duration noted is three years; the longest, thirty-six years; the average duration, fifteen years. In one case the disease lasted three years; in two four years; in two, thirteen years; in two, nineteen years; in one twenty years; in one, twenty-five years; in one thirty-six years.

But few died of the disease itself; the majority usually die of intercurrent diseases:

Patient 1 died of tuberculosis of the lung and peritoneum; Patient 2, after splenectomy; Patient 3, of asthenia due to the disease itself; Patient 4, of asthenia due to the disease itself; Patient 7 died three hours after splenectomy; Patient 9, of disease (pericarditis); Patient 10, of accident; fracture of skull; Patient 11, of septic cholangitis. Patient 14 died suddenly. Patients 3, 15 and 16 were still living at the time of this report.

DIAGNOSIS

The disease which splenomegaly (Gaucher) resembles most, but from which it differs greatly, is anemia splenica. The differential points will be noted from the following:

ANEMIA SPLENICA	SPLENOMEGALY (GAUCHER)
Family Incidence: Not more than one in a family	Three families with two cases; two families with three cases.
Sex: Males, 39 out of 47.	Females: 12 out of 16. 3-13; 30-41.
Age: Low limit (9-20) High limit (58-72).	
Pathology: Spleen shows a hyperplastic fibrosis, involving particularly the Malpighian bodies. The liver shows no characteristic changes. The lymph-glands are not enlarged and show no changes. The bone marrow shows compensatory changes, secondary to secondary anemia.	Spleen, liver, lymph-glands and bone-marrow show endothelial hyperplasia.
Chronicity: Four to ten years.	Three to thirty-six years.
Splenomegaly: Large.	Larger.
Size of smallest spleen, 790 gm.	Smallest, 880 gm.
Size of largest spleen, 5,670 gm.	Largest, 7,000 gm.
Average of fifteen cases, 1,883 gm.	Average of nine cases, 4,300 gm.
Anemia: Secondary type.	Simple anemia.
Average red blood count, 3,425,000	Average, 4,000,000 reds; 5,600 whites; Hgb. 70 per cent.
Average white blood count, 3,850	
Average Hgb. estimation, 47 per cent.	
Hemorrhages: As a rule, hematemesis in one-half the cases; severe.	Never hematemesis, but epistaxis and oozing from the gums.
Ascites and edema: Occasionally seen.	Never.
Jaundice: Moderate in degree; veins in association with cirrhosis of the liver.	Never.
Liver: Is of normal size in most cases.	Greatly enlarged in a majority of the cases.

BANTI'S DISEASE	SPLENOMEGALY (GAUCHER)
Sex: Females 32 out of 50 cases.	Females 12 out of 16 cases.
Heredity: Not congenital; does not affect more than one member of the same family.	Is probably congenital; usually affects more than one member of the same family.
Age: Youngest of 50 patients is 12 years; oldest of 50 patients is 55 years.	Youngest recorded is 3 years; oldest recorded is 41 years.
Spleen much enlarged.	Larger.
Liver at first of normal size; then becomes enlarged, and later diminishes in size.	Always enlarged.
Jaundice sometimes present.	Never present.
Ascites usually present.	Never present.
Blood: R. B. C., 4,330,000; W. B. C., 3,800; Hgb., 56 per cent.	R. B. C., 4,200,000; W. B. C., 6,000; Hgb., 70 per cent.
Hemorrhages: Usually severe, in the form of hematemesis.	Never severe; usually epistaxis or oozing from the gums.
Duration seven to ten years.	Three to thirty-six years.
Death: Usually due to intoxication from cirrhosis of liver or to severe hemorrhage.	Usually due to intercurrent disease.
Pathology: There is a fibrosis of the reticulum of the spleen, the liver and the portal vein. Bone marrow and lymph-glands are normal.	There is an endothelial hyperplasia of the spleen, the liver, the lymph-glands and the bone marrow.

In children it is most important to differentiate splenomegaly (Gaucher) from von Jaksch's disease (anemia pseudoleukemica, or according to more recent nomenclature, anemia splenica infantum). Differential points are as follows:

SPLENOMEGALY (GAUCHER)	VON JAKSCH'S DISEASE
1. Always primary.	1. Usually secondary to gastro-intestinal disease, malnutrition, tuberculosis, lues, etc.
2. Usually affects children between the ages of 2 and 4 years.	2. Affects children between 6 months and 2 years.
3. Discoloration usually yellow or orange; more marked in some parts.	3. No discoloration, but there is extreme pallor of whole face, which may be olive color.
4. Spleen much larger; weighs 1,500 gm.	4. Spleen enlarged but not to the same extent seen in S. (Gaucher). Spleen weighs 350 gm.
5. Liver usually enlarged.	5. Liver usually not enlarged.
6. Glands (lymph) never enlarged.	6. Lymph-glands sometimes enlarged.
7. Blood: Red blood corpuscles, 4,100,000; white blood corpuscles, 5,600; Hgb., 70 per cent.	7. Blood: Red blood corpuscles, 2,500,000 to 3,000,000; white blood corpuscles, 10,000 to 20,000; Hgb., 45 per cent.
No abnormal cells found.	Myelocytes and normoblasts in 25 per cent of the cases.

Some of the other conditions from which splenomegaly (Gaucher) should be differentiated are: pernicious anemia, polycythemia, leukemia, aleukemic leukemia, cirrhosis post leukemia, status thymicus, cirrhosis post myxedema, abscess of spleen, lues, tuberculosis, malaria, etc., and the cirrhosis due to different causes, as alcoholic, hematogenous, cardiac,

metabolic, chromatic, adenomatosa, and that due to tuberculosis, distoma and the infections.

SUMMARY

1. Splenomegaly (Gaucher) is a distinct disease.

2. It is probably a congenital condition, and usually affects more than one member of the same family. Females are more susceptible to this disease than are males (3 to 1).

3. The first symptoms may appear as early as the third year or as late as the twentieth year.

4. The spleen becomes enormously enlarged and is followed by an enlargement of the liver (but the enlargement of the liver is not as great as occurs in the spleen).

5. Vague abdominal pains are present. There is pigmentation of the face and hands, the color being brown, yellow or bronze.

6. Hemorrhages, if present, are not severe; usually are in the form of epistaxis or oozing from the gums.

7. The disease is slowly progressive and is marked by periods of temporary improvement. The disease may last from three to thirty-six years.

8. The majority usually die of intercurrent diseases; but few die of the disease itself.

9. At autopsy there is found an endothelial hyperplasia in the spleen, liver, lymph-nodes and bone-marrow.

In conclusion I wish to express my indebtedness to Dr. Herrman, by whose courtesy I am enabled to report the cases of the clinic. I also wish to express my thanks to Dr. E. Cleaver, who performed the blood examinations on the patients here reported.

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THE TREATMENT OF PERTUSSIS WITH VACCINE*

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The seriousness of pertussis in the infant, its tendency to be complicated by pneumonia, emphysema, convulsions and perhaps paralysis, and the possibility of a fatal termination, have caused this disease to occupy a prominent place in the minds of all physicians, particularly as it is so commonly seen in infancy, 50 per cent. of all cases occurring in the first two years of life. As the treatment of pertussis by drugs is so unsatisfactory, it is important that any new treatment that offers a reasonable hope of success should be thoroughly tested.

Pertussis is undoubtedly contagious and positively due to a micro-organism, and it seems to me that the bacillus described by Bordet and Gay in "Studies in Immunity" has many claims to the position of the specific pathogenic organism.

Bordet and Gengou[1] claim to have found the true bacillus of pertussis. They assert that the bacillus is found only in the early period of the disease, and only that part of the expectoration which comes from the region in which the bacteria are most active should be used; i. e., "the products from the depths of the bronchi, expectorated during the periodic accesses of the disease." This exudate contains the true bacillus in considerable numbers and, in favorable cases, in almost pure culture. "As the disease progresses, on successive days fewer and fewer specific bacilli are found and phagocytosis is more frequent than earlier." They emphasize the importance of studying cases not complicated with other respiratory affections, and of avoiding the study of the sputum of hospital patients, as in the latter other organisms are apt to be present in great numbers.

They describe the micro-organism of pertussis as "little ovoid bacilli, somewhat elongated at times, but frequently so short as to resemble a micrococcus." With Kuhne's carbolated blue stain, the bacilli will stain a pale blue, and the outline of the bacillus, and particularly its poles, are frequently more deeply stained than the center. "This organism was found scattered indiscriminately among the cells and was at times within cells." It was negative to Gram's stain.

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*Read before American Pediatric Society, Lake Mohonk, June 2, 1911.

1. Bordet and Gengou: Studies in Immunity, 1906, chap. xxvii.

This bacillus does not grow well on blood-agar. It develops well in a liquid medium of 1 per cent. glycerinated bouillon with equal parts of rabbit's blood or serum. It is found that the organism grows only under the best condition of aerobiosis.

"The authenticity of this bacillus as the causal agent in whooping-cough rests in large part on the circumstances in which it is found, namely, the excessive growth of the organism in pure culture during the initial period of the disease in young children who have never been ill. The principal argument, however, in favor of this organism appears to be furnished by a study of the specific properties of the serum. The serum of individuals who have never had whooping-cough, or who have had it a long time before, does not agglutinate the bacillus even in low dilution. The serum of children who have recently recovered from the disease has a moderate agglutinating property which is constant and evident. The most remarkable fact, however, is the intensity of the sensitizing property in such serums."

Bordet and Gengou assert that the organism described by Pfeiffer resembling the influenza bacillus, "acts no differently with whooping-cough serum than it does with normal serum." They find that the serum of children suffering from, or convalescent from whooping-cough, shows very inconstant agglutinating properties. An agglutinating property in a mild form is frequently present; it may be entirely lacking. They find, however, that the alexin fixation method always gives marked positive results.

Bordet, as described in a later communication (1908), has obtained from the organism an endotoxin. He and his co-worker hope to obtain an antitoxic serum by injecting endotoxin instead of bacteria, and claim that the bacillus produces a poison that causes a necrosis of that portion of the epithelium covering the bronchi where rapid increase in the number of the bacilli occur, and that this necrosis is the cause of the characteristic symptoms of pertussis and their persistence.

As I was without any previous knowledge of the amount of vaccine to administer and was consequently ignorant as to the number of bacteria to inject, my earliest patients received only 20 million bacteria every four days. This dose failed to produce any untoward symptoms locally or constitutionally, and was increased to 40 million bacteria every four days. A careful study of the patients receiving this dose, by all those coming in contact with the patients, including parents, other physicians and myself, tended to show that the disease was apparently being modified by the treatment. The same dose was then given every three days, and the beneficial results seemed to be distinct. The number of paroxysms of coughing became less; the severity of the paroxysms diminished, cyanosis was less marked, vomiting during the coughing was less frequent, and the children, as a rule, slept better and were less disturbed at night, since the number of characteristic attacks became less. The dose of 40 million was retained, but the injection was now given every two days. The num-

ber of injections given varied from six to eight. No case has been recorded in which less than six doses were administered, and none of the patients received more than eight injections of 40 million each, except twenty-two, who had nine injections. In not a single instance were evidences of local inflammation noticed at the site of the injections, and while most of the injections were given in the loose tissues of the scapular region, some children received injections into the arms.

The interscapular region is, in my judgment, the best place for injection, as it is difficult for the child to scratch this portion of the body, and it is apt to be fairly well protected by clothing. Care was taken to render the part injected clean with soap and water and alcohol and to sterilize the hypodermic syringe. Neither I nor any other physician to whom I gave the vaccine noticed any local or constitutional effects from the remedy; rashes, joint pains and fever were carefully looked for, but were never observed.

All but one of the cases were in private practice, and this affords a much better test of the efficacy of the remedy than a study of hospital cases. Institution children in whom whooping-cough develops while in the ward, are immeditely sent to the pertussis pavilion, and are apt, therefore, to be in intimate contact with other pertussis patients, who may have other infections of the respiratory organs. Moreover, institution children do not, as a rule, do well when attacked by infections, and are therefore less apt to show the true beneficial effects of which a remedy is capable. I do not wish to be understood as suggesting that it should not be used on hospital patients, but they are manifestly less apt to exhibit the true influence of the remedy, as they are, as a class, less vigorous and robust than children living outside of institutions. This, of course, applies more to children under 2 years of age. Older children are less apt to be affected with hospitalism. Outside children admitted to hospitals with pertussis are apt to be received late in the disease and commonly have some complication, often pneumonia. I would strongly advise that all these children be given the benefit of the vaccine treatment; I believe that a thorough trial of it in these cases will demonstrate its efficacy; but for the purpose of this investigation and study, private cases are better.

Physicians are apt to be too optimistc in ascribing to new drugs or methods of treatment curative properties. A more extended trial may prove their conclusions to be premature and unfounded, and this may be the fate of pertussis vaccine. In order at least partially to eliminate this error, those coming in intimate, daily contact with the patients, parents, nurses and other members of the household, have been systematically questioned, and their opinions, in the large majority of cases, have been in favor of the remedy—that it tends to diminish the severity and number of the attacks and shorten the course of the disease.

After the injections were stopped, the cases were followed as closely as possible to see whether the usual course of the disease was influenced only during the use of the remedy, and whether the paroxysms of coughing would return after the discontinuance of the vaccine. This method of following the cases resulted in showing that improvement almost invariably continues steadily after the course of treatment by the vaccine has been discontinued.

It is, of course, well known that epidemics of pertussis differ greatly in their severity, and it may be that future cases will be less influenced by the treatment than those cases on which this paper is based. I have seen personally many of these cases, and in my judgment they were cases of ordinary severity. In the list of cases here reported, no child was included who did not have the typical whoop, and this typical whoop was heard by the attending physician in each instance. The judgment of the parents or nurse was never taken on this point.

In all, twenty-four cases of pertussis are reported. The ages of the children were as follows:

	Cases
Less than 6 months	3
6 months to 1 year	2
1 to 2 years	4
2 to 4 years	10
4 to 8 years	5

REPORT OF CASES

CASE 1.—Mary H., aged 3 years, colored, pertussis ward, Philadelphia Hospital; first injection given March 14, 1911; whooping two weeks before treatment began; received six injections, three of twenty million, three of forty million. A careful systematic daily record was kept of the number of the paroxysms in every twenty-four hours, of the duration in minutes and seconds of each paroxysm, whether severe, moderate or mild, and the frequency of vomiting produced by the paroxysms of coughing. Careful records of the temperature, pulse and respiration were recorded only in those cases in which there was fever. The record shows that the child was much benefited by the treatment. The paroxysms were reduced from an average of twenty in twenty-four hours to five in twenty-four hours. The duration of the paroxysms was much shortened. They were less in severity, and the frequency of the vomiting attacks diminished. The temperature, respiration and pulse all improved. From March 14 to 20 the temperature varied from 101.5 to 100 F.; March 21 to 24, from 101 to 100 F.; March 24 to 28, 101 to 100 F.; March 28 to April 1, 100 to 99 F.; April 1 to April 8, normal.

There is no doubt in my mind that the child was distinctly benefited by the treatment. The records kept in the hospital are too voluminous to publish. However, the portion of the record here appended is of interest.

HOSPITAL RECORD OF MARY H.

Admitted to whooping cough isolation ward March 1, 1911. Received vaccine lymph (pertussin), 20,000,000, March 14, 1911.

First dose March 14: March 14, 4 p. m., to March 15, 4 p. m., total number of times coughed, 18; coughed hard 14 times; vomited, 7 times; coughed not so hard, 4 times; expectorated, 7 times.

March 15, 4 p. m., to March 16, 4 p. m., total number of times coughed, 12; coughed hard 5 times and vomited; coughed hard 7 times and expectorated.

March 16, 4 p. m., to March 17, 4 p. m., total number of times coughed, 11; coughed not so hard, 4 times; coughed hard, 4 times; coughed hard and vomited, 3 times.

Second dose, March 17: March 17, 4 p. m., to March 18, 4 p. m., total number of times coughed, 10; coughed not so hard, 4 times; coughed hard, 3 times; coughed hard and vomited, 3 times.

March 18, 4 p. m., to March 19, 4 p. m., total number of times coughed, 13; coughed hard and vomited, 6 times; coughed hard, 7 times.

March 19, 4 p. m., to March 20, 4 p. m., total number of times coughed, 11; coughed hard and vomited, 5 times; coughed hard, 6 times.

Third dose March 20: March 20, 4 p. m., to March 21, 4 p. m., total number of times coughed, 8; coughed not hard, 4 times; expectorated; coughed hard, 4 times, vomited.

March 21, 4 p. m., to March 22, 4 p. m., total number of times coughed, 8; coughed not hard, 4 times; expectorated; coughed hard, 4 times; vomited.

March 22, 4 p. m., to March 23, 4 p. m., total number of times coughed, 9; coughed not so hard, 4 times; coughed hard, 5 times; vomited, 4 times.

Fourth Dose, March 23: March 23, 4 p. m., to March 24, 4 p. m., total number of times coughed, 8; coughed not hard, 3 times; coughed hard, 5 times; vomited 3 times.

March 24, 4 p. m., to March 25, 4 p. m., total number of times coughed, 6; coughed not hard, 2 times; coughed hard, 4 times; vomited 3 times.

March 25, 4 p. m., to March 26, 4 p. m., total number of times coughed, 6; coughed not hard, 1 time; coughed hard, 5 times; vomited 3 times.

Fifth Dose, March 26: March 26, 4 p. m., to March 27, 4 p. m., total number of times coughed, 6; coughed not hard, 3 times; expectorated; coughed hard, 3 times, vomited.

March 27, 4 p. m., to March 28, 4 p. m., total number of times coughed, 6; coughed not so hard, 3 times; coughed hard, 3 times; vomited 2 times.

March 28, 4 p. m., to March 29, 4 p. m., total number of times coughed, 5; coughed not so hard, 1 time; coughed hard, 4 times; expectorated 4 times; vomited 1 time.

Sixth Dose, March 29: March 29, 4 p. m., to March 30, 4 p. m., total number of times coughed, 5; coughed not hard, 2 times; coughed hard, 3 times; vomited 2 times; expectorated 3 times.

March 30, 4 p. m., to March 31, 4 p. m., total number of times coughed, 5; coughed not hard, 1 time; coughed hard, 4 times; expectorated 3 times; vomited 2 times.

March 31, 4 p. m., to April 1, 4 p. m., total number of times coughed, 8; coughed not hard, 3 times; coughed hard, 5 times; vomited 3 times.

April 1, 4 p. m., to April 2, 4 p. m., total number of times coughed, 6: coughed not hard, 1 time; coughed hard, 5 times; vomited 3 times.

CASE 2.—Elizabeth H., aged 3 years and 10 months; report by Dr. E. D. Grier; treatment began April 11, 1911. The child had been coughing and whooping several weeks before the first dose of pertussis vaccine was given. She received five injections of twenty million and three of forty million each. The attack was of moderate severity. The child was much improved at the end of the treatment. The cough was almost gone and there was very little whoop.

The following is a brief résumé of the notes kept by the mother, a very intelligent woman.

April 11, 1911.—From 6 to 12:15 a. m. had coughed ten times; vaccine given at 12:15; coughed five times from 12:15 until 8 p. m.; vomited twice slightly.

April 12, 1911.—Coughed once during the night; coughed four times from 6 a. m. until 12 M.; vomited once; coughed three times from 12 M. until 6 p. m.; in all eight paroxysms.

April 13, 1911.—Coughed twice during the night; coughed four times during the morning, and four times during the afternoon; no vomiting.

April 14, 1911.—Coughed once during the night; no vomiting; coughed seven times during the day; appetite good.

April 15, 1911.—Coughed once during the night, about 5 o'clock; coughed seven times during the day; vomited once after taking 10 ounces of milk. Dr. Grier administered vaccine at 10 a. m.

April 16, 1911.—Coughed once during the night about 5 o'clock, but not since 4:30 the previous evening; seven times during day. Vomited once slightly.

April 17, 1911.—Coughed six times; vomited most of the first bottle of milk at 6:30 a. m., but ate a good breakfast. She takes a nap every morning of 1½ hours; seems much improved. The worst coughing spell is in the morning when she has some trouble in bringing up the phlegm.

April 18, 1911.—Coughed early in the morning; seven times during the day, and vomited a very small amount in the afternoon. "Severe cough at 3:30 p. m., while I was present."—Grier.

The baby received the last dose of vaccine May 6, and under this date Dr. Grier writes me as follows: "The family feel that the child is much improved, and the attack I heard to-day bears out their statement, as it was of no moment; the cough now is almost nothing."

CASES 3 AND 4.—Edwin W. and Kenneth W., brothers, aged 7 years and 4 years; first injection of pertussis vaccine April 11, 1911. Each received eight injections of forty million. Both had been whooping about one week before the treatment began. Both were benefited by treatment, the older boy more than the younger. In both children the number and severity of the paroxysms diminished. The younger boy had adenoids and tonsils removed May 20, six weeks after the beginning of the vaccine treatment. He was so much better that we did not hesitate to place him in a private room in a hospital for the removal of his adenoids and tonsils.

CASE 5.—William N., aged 15 months; first injection of pertussis vaccine May 10, 1911; whooping five days before treatment began; received eight injections of forty million each. After the sixth injection the child had a slight bronchopneumonia; temperature varied from 101 to 103 F. for five days. The vaccine treatment apparently did not affect the pertussis in any degree whatever. The patient in my opinion was not benefited by the treatment. At the end of the treatment the boy was still coughing and whooping, but neither the cough nor the whoop was severe in character and the boy's temperature was normal. I believe that the dose in this case was insufficient to produce an effect. Instead of forty million, he should have been given at each dose eighty to 100 million.

CASE 6.—Francis N., 2½ years old, sister of Patient 5; first injection of pertussis vaccine May 10, 1911; whooping two weeks when treatment began. A severe case; numerous paroxysms day and night; vomited frequently, and often during attack became cyanosed; eight injections of forty million each were given. The paroxysms of coughing after the child had received four injections were distinctly less severe, although not diminished in number. After receiving the full number of injections, eight, the boy was much improved. The paroxysms were few in number, and very light, with only an occasional whoop.

CASE 7.—Isabella L., aged 4 months; whooping two weeks before treatment began; eight injections of forty million each were given; much improved; very little cough and occasional whoop. This child had a mild attack of pertussis, and at the end of treatment was practically well. Very little cough remained and hardly enough whoop to designate it by that name.

CASE 8.—David L., aged 2 years; first injection of pertussis vaccine May 12, 1911; whooping one week before treatment began; seven injections of forty million each were given. A careful record of the number and severity of the attacks was made. In the early part of the treatment the attacks were very severe; thirty-six in twenty-four hours. At the end of the treatment the attacks averaged

about twenty-eight in twenty-four hours. There was no decrease in the severity of the attacks. The patient was apparently not benefited by the treatment. The case was reported by Dr. W. S. Lucas. I believe the dose of vaccine used in this case was too small. In a similar case I would advise eighty million or 100 million with each injection.

CASE 9.—Charles L., aged 8 years, brother of Patient 8; first injection of pertussis vaccine May 10, 1911; whooping ten days before treatment began. This was a much milder case than that of his brother, David L., Case 8. Patient 9 received eight injections of forty million each. At the beginning of treatment, he averaged fifteen paroxysms in twenty-four hours, moderately severe in type, about five or six attacks occurring at night. At the end of treatment he averaged about six or seven attacks in twenty-four hours, very mild in character, and not more than one or two at night. The boy was evidently much improved by treatment. Reported by Dr. W. S. Lucas.

CASE 10.—Fanny C., colored, aged 5 months; first injection of pertussis vaccine given May 5, 1911; a sturdy child, breast-fed, always in good health until the attack of pertussis. Paroxysms occurred every hour during the day and three or four times at night, vomiting with almost every paroxysm; face became deeply congested with each attack of coughing. Six injections of forty million each were given; there was so much improvement at the end of the sixth injection that the mother considered the child well and wished the treatment discontinued. The child still coughed and whooped occasionally, but the paroxysms were very mild.

CASE 11.—Male, aged 5½ years; reported by Dr. S. M. Wilson; whooping one week before treatment began; eight injections—three of twenty million, four of forty million and one of sixty million; first injection April 29, 1911. Dr. Wilson wrote me as follows: "At the time of his first injection, he had twenty or more severe paroxysms daily and his sleep at night was badly disturbed by them. At the end of the treatment he rarely coughs, and when he does, there is usually only a single effort and no expectoration." Dr. Wilson considered that the boy was very much benefited by the treatment.

CASE 12.—Cecil D., aged 3 years; first injection of pertussis vaccine May 5, 1911; reported by Dr. H. W. Gregory. The child had been whooping three weeks before treatment began. The attacks were severe, occasionally producing hemoptysis; eight injections of forty million each were given. At the end of the treatment the child was practically well, averaging only one mild paroxysm during the night and with very little cough during the day.

Cases 13, 14, 15, 16 and 17 all occurred in the practice of Dr. S. M. Cleveland, and according to Dr. Cleveland's observations the patients were apparently not benefited by the treatment. The only explanation that I am able to give for the failure of the treatment in these cases is that the dose of vaccine used was not large enough. A brief history of these cases follows:

CASE 13.—Carl O., 16 months old, whooping five days when treatment began; seven injections of forty million each produced apparently no benefit.

CASE 14.—Dorothy O., sister of Patient 13, aged 4 months; whooping seven days; given seven injections forty million each; apparently no benefit from treatment.

CASE 15.—Elizabeth O. B., 10 months old, sister of Patient 16; coughing two months before treatment began; six injections of forty million each were given; apparently no benefit resulted from treatment.

CASE 16.—Marie O. B., aged 5 years; coughing for two months before treatment began; six injections of forty million each were given, with no benefit.

CASE 17.—James O. B., aged 7 years, brother to Patients 15 and 16; coughing two months before treatment began; six injections of forty million each were given, with no benefit.

CASE 18.—James L., aged 28 months; first injection of pertussis vaccine given May 9, 1911; whooping three weeks when treatment began; eight injections of forty million each were given. The patient was a delicate boy and was having paroxysms hourly day and night, with occasional vomiting. The patient began to improve after the first injection; vomiting ceased after the second injection. At the end of the treatment there was a little cough, but no whoop. Reported by Dr. John A. McCormick.

CASE 19.—John L., aged 2 months, brother of Patient 18, was given the first injection of pertussis vaccine May 9, 1911; whooping three weeks before treatment began; eight injections of forty million each were given. The case was moderate in severity, and the patient was apparently much benefited by treatment. At the end of the treatment the cough occurred less frequently; the paroxysms were mild. Reported by Dr. John A. McCormick.

CASE 20.—Genevieve B., aged 15 months; treatment began May 8, 1911, after whooping had existed four weeks. The first injection of pertussis vaccine was on May 8; eight injections of forty million each were given. At the beginning of treatment the child had about sixteen paroxysms in twenty-four hours, ten during the day and six during the night. The paroxysms were very severe, with frequent vomiting and cyanosis; improvement began after the third injection; distinct improvement was noticed after the fifth injection. At the end of treatment the child was still coughing, but only an occasional whoop. Reported by Dr. John A. McCormick.

CASE 21.—John B., aged 3 years, brother of Patient 20; first injection of pertussis vaccine May 8, 1911; whooping three weeks when treatment began; eight injections of forty million each were given. The case was of more than ordinary severity. The paroxysms occurred about once an hour day and night, with cyanosis and vomiting. The cyanosis and vomiting disappeared after the fourth injection. After the fifth injection, the mother thought the child was well enough to stop the treatment; eight injections, however, were given. At the end of the treatment, the child was almost entirely well, with practically no cough and no whoop. Reported by Dr. John A. McCormick.

CASE 22.—Edward S., aged three years, treatment began May 8, 1911. Hemorrhage into the conjunctiva occurred as the result of the severity of the paroxysms. No improvement was noticed until after the fourth injection. Vomiting ceased after the sixth injection. The patient received nine injections of forty million each. At the end of the treatment the boy was very much improved, although he still coughed and whooped occasionally. Reported by Dr. John A. McCormick.

CASE 23.—Emma M., aged 9 months; first injection of pertussis vaccine May 6, 1911. Whooping commenced one week before treatment began. Seven injections of forty million each were given. Cyanosis and vomiting were frequent at the beginning of treatment. Paroxysms occurred at least every hour during the day and five or six times during the night. At the end of the treatment the paroxysms were few in number and less severe, but the child was still coughing and whooping occasionally. Reported by Dr. Scott Lau.

CASE 24.—William B., aged 5 years; first injection of pertussis vaccine May 8, 1911; whooping eleven days before treatment began; eight injections of forty million each were given. The case was one of moderate severity. At the end of treatment the paroxysms of coughing were much fewer in number and less severe, although the boy still coughed and whooped occasionally. He was distinctly benefited by the treatment. Reported by Dr. Scott Lau.

These reports do not represent the true worth of the remedy, as I believe better results would surely have been obtained had larger doses of the vaccine been employed; 40 million may be, and probably is, a sufficient dose for a mild case in an infant or young child. One hundred million

would be a much more suitable dose for a severe case, regardless of the age of the patient.

Of the twenty-four reported cases, seven were apparently not benefited by the treatment, the disease evidently pursuing its usual course in about 29 per cent. Seventeen patients, or about 71 per cent., were apparently benefited by the treatment. I believe the dose could, and should, be increased beyond 40 million, especially for severe cases; 60, 80 or 100 million will probably be a more efficient dose. The results obtained in these twenty-four cases warrants, in my opinion, a more extensive trial of the vaccine.

RESUSCITATION OF ASPHYXIATED INFANTS BY THE INSUFFLATION METHOD OF MELTZER AND AUER

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The remarkable experiments of Meltzer and Auer on intratracheal insufflation were made known in France by Carrel, who also brought them to my attention. I was greatly impressed by them and concluded that the principle might be applicable to the resuscitation of asphyxiated infants, for which purpose I had an apparatus constructed by Mr. Lépine of Lyons. The apparatus is very simple and resembles the one used on animals at the Rockefeller Institute and differs only in being of a size suitable to new-born infants.

I have employed this apparatus and the method of intratracheal insufflation in my obstetrical service since August, 1910, with promising and highly interesting results. I make this statement because I am of the opinion that it will require several years' experience to determine its exact clinical value and extent of applicability. I am, however, ready to affirm that the principle of the method embodies a great advance and its efficiency is already beyond question for the reanimation of asphyxiated infants.

The principle of the method of Meltzer and Auer is well known. A continuous current of air directed as far as the bifurcation of the trachea through a tube of small caliber, produces a ventilation of the lungs sufficient for oxygenation of the blood even if there are no respiratory movements. The air injected into the bronchi returns between the catheter and the tracheal wall, and can also remove foreign bodies and mucus that may be in the respiratory tubes. The apparatus constructed by Lépine is composed of:

1. A rubber bulb.

2. A T tube, one branch of which is united to the bulb, and another to a small mercury manometer, while the third is connected to a rubber tube ending in a metal cone.

3. A gum catheter No. 12. A rod of soft copper is placed in the catheter in order to give it the proper shape for introduction into the glottis. The caliber of the catheter was selected after an anatomic study of the trachea on the cadaver. The internal diameter of the trachea of a new-born child is 4 mm. There is allowed, therefore, between the wall of the trachea and a catheter No. 12 (Charrière) a sufficient space for the

returning current of air. The end of the catheter must reach the bifurcation of the trachea. This point is located at a distance from the mouth which varies according to the size of the child. For a child weighing 2,000 gm. the distance is 8 cm.; 3,000 gm., 10 cm.; 4,000 gm., 12 cm. Therefore, three transverse lines are marked on the catheter to indicate how far it should be introduced according to the weight of the child.

OPERATIVE PROCEDURE

The mucus contained in the throat of the infant in a condition of asphyxia is rapidly removed by the finger or by a little sponge fixed in forceps. The infant is wrapped in a warm blanket and placed on a table, the neck being in slight hyperextension.

The index finger of the left hand of the operator is introduced as far as the upper end of the esophagus so as to feel the hard rounded prominence which is formed by the posterior face of the larynx, the two arytenoid cartilages, and to find in front of them a soft opening, which is the glottis. The catheter, containing its copper rod, to which a proper curve has been given, is introduced by the right hand between the tongue of the child and the palmar face of the left index finger of the operator and thus penetrates easily into the trachea.

The catheterization is exceedingly easy. After a few trials on the cadaver, I could always do it without trouble. Moreover, the interns, externs, and nurses of my service succeeded also in introducing the catheter very easily. The average nurse can certainly acquire the technic without difficulty.

When the catheter has reached the proper distance, the copper rod is removed. The end of the insufflation apparatus is now attached to the catheter and air is injected through the catheter by the rubber bulb, while the mercury manometer is watched. The pressure must not exceed 10 or 15 mm. Soon the noise of the returning current of air passing through the lips of the child is heard. The manometer indicates that there is no over-pressure by accumulation of air in the respiratory apparatus. Therefore, the conditions of ventilation are known, and no accident is to be feared.

The insufflation is continued as long as is necessary. After a few minutes, the child appears to be less atonic, the heart becomes stronger and more regular, and the respiratory movements start. The catheter is removed when the infant is in a condition to carry on automatic respiratory movements.

CLINICAL RESULTS

The method must be applied under well-defined conditions. If it is used in mild cases of asphyxiation, it is certain that it will give constant positive results as do all the other methods. If it is used in the cases in

which apparent death is not due to asphyxiation but to traumatism of the brain or of the medulla oblongata, it will yield negative results. Therefore, the value of the method must be ascertained in the cases of deep asphyxia of the new-born by long intra-uterine compression or by interference with the fetal-maternal circulation, as, for instance, from compression of the umbilical cord.

I have employed the method in seven cases of the last kind. The seven infants were restored to life after a longer or shorter period of insufflation, although two of them had been extricated after difficult versions.

Observation 1.—Child weighing 2,760 gm.; Braxton Hicks version for placenta prævia; no respiratory movements; heart regular, but very slow; insufflation for twelve minutes; afterward, movements of the face and respiratory movements. The child cried as the catheter was removed.

Observation 2.—Child weighing 4,200 gm.; difficult version, with prolonged extraction of the head; asphyxia pallida; irregular heart; a few diaphragmatic convulsions; very infrequent. Reanimation occurred after insufflation during eight minutes.

Observation 3.—Child weighing 3,600 gm.; extracted by forceps; probable prolapsus of cord; child was atonic, without respiratory movements; meconium evacuated; heart hardly perceptible. Reanimation occurred after insufflation during seven minutes.

Observation 4.—Child in a condition of blue asphyxia, but the heart was almost normal. The child was quickly reanimated.

Observation 5.—Similar to Case 4. It is probable that in these two cases the reanimation would also have taken place easily if the ordinary methods had been employed.

Observation 6.—Child weighing 1,970 gm.; premature labor; high forceps delivery; asphyxia pallida; no respiratory movements; very irregular and slow heart; grave form of apparent death. After five minutes of insufflation the heart became regular; reanimation after eight minutes.

Observation 7.—Child weighing 3,000 gm.; long labor; period of expulsion lasted five hours; blue asphyxia. Reanimation occurred after insufflation for six minutes.

The method deserves to be employed also in cases of traumatism to the skull and brain. I am of the opinion that it will excel other methods in this condition, although it will, of course, be limited by the severity of the injury, which may be incompatible with life.

PROGRESS IN PEDIATRICS

ANATOMY, PHYSIOLOGY AND HYGIENE

Milk-Station Work and the Reduction of Infant Mortality

(Godfrey R. Pisek: New York Med. Jour., Nov. 25, 1911, p. 1065)

During 1911, New York City conducted a campaign against infant mortality through milk stations sufficiently comprehensive to show definite results. New York has 125,000 births yearly. Various private philanthropies have been dispensing modified milk to indigent mothers for some years. In 1911, the Committee for the Reduction of Infant Mortality established thirty-one milk depots and in addition secured an appropriation for the establishment of fifteen stations under the control of the health department, making seventy-nine stations in all. To secure cooperation of all the different agencies, the Association of Infants' Milk Stations was formed in June, 1911, and 74,000 quarts of milk were distributed weekly. The work was not confined to the distribution of milk, however, Education of the mothers was the most important factor. The intimate contact of the visiting nurse with the mothers in their homes made it possible to teach the modification of milk according to the doctor's instructions. In this work whole milk was used for the most part, modified in many instances with gruels and ordinary granulated sugar, which was found satisfactory. Deterioration in the homes was prevented by the use of inexpensive home-made refrigerators. Mothers were encouraged to continue breast-feeding where this was possible. Special diet lists were prepared for older children. Another feature of the work was the assistance and advice given in regard to protecting from infectious diseases and ophthalmia. The Association issued weekly bulletins giving comparative statistics of infant mortality in different cities. Three hundred and fifty-five cities cooperated in this. The results of the work in New York in figures were as follows: The number of deaths of infants from all causes under one year of age during the first nine months of 1911, was 11,733; while during a corresponding period of 1910 the deaths reached 12,920; in other words, there was a comparative death-rate of 124.6 in 1911, against 142.3 in 1910, a decrease of 17.7 per thousand among infants. This rate continued throughout the remainder of the year would result in a saving of 1,640 babies during the year.

Even more striking than the statement that 1,187 fewer deaths occurred up to October 1 compared to 1910, is the fact that during the months of July, August and September there were 1,244 fewer deaths than the previous year. To show that this comparison is not made with an excessively high year, it might be mentioned that there had been a decrease of 1,417 deaths from the average for these months during the five years 1906 to 1910.

In the Borough of Manhattan, from January to June, there was an increase of deaths from diarrheal diseases of 28 per cent. over the previous year, but during the month of June, after beginning the milk campaign, there was a decrease of 50 per cent. over the previous year. In July, which was exceedingly hot, there was a decrease of 60 per cent., August 23 per cent., September 25 per cent., the average decrease being 41 per cent. Comparing these results with those in other cities reported on, it was found that the diminished mortality was in direct relation to the effective preventive measures employed. Comparison between districts in the congested quarters, with and without milk stations, showed a gain over the previous year of 29 per cent. in the milk-station districts as against an increase of 9 per cent. in those without milk stations. It is felt that the best feature of the work is the educational feature.

Suggestions for the Reduction of Infant Mortality

(*S. Josephine Baker: New York Med. Jour., Nov. 25, 1911, p. 1067*)

The infant mortality rate is the index of the sanitary progress of any community. No one line of endeavor, however vigorously worked out, will permanently lower the death-rate, and Baker believes there is serious danger in assuming that total reduction in death-rate in any one year is wholly due to the specific efforts made during that year. The keeping of babies well cannot be worked out with the accuracy of a scientific theorem, but the fundamental principle is that prevention of diseases must be the aim and education the main weapon. Specifically, the principles of the reduction of infant mortality include:

1. The study of the problem of the institution baby. During 1911 up to October 1, 42 per cent. of all deaths of babies under one year in the Borough of Manhattan occurred in institutions, the foundling babies furnishing the greater proportion.

2. A supply of milk for infant feeding at a price within the reach of the majority of people. The relative value of raw and pasteurized milk for infant feeding still seems an open question. Purification cannot take the place of purity, but the matter is economic, and purity seems beyond our reach at the price we can pay.

3. The broadening of courses in pediatrics in our medical colleges, so that they may assume the importance that is their due and assure to every physician a thorough knowledge of infant hygiene and care.

4. The interest and attention of social students and workers, and of philanthropists, in meeting individual family needs and adjusting economic conditions.

5. Instruction of each mother, first in the necessity of nursing her baby, and, if that is impossible, then in the proper substitute feeding, and how she may take advantage of and apply the essential methods of hygienic baby care.

6. A right understanding of the immediate causes of infant mortality.

Baker then enters into the statistics of 1911 as given in an abstract of a paper by Pisek in this issue. The figures show what may be accomplished by the application of the principles enumerated above but Baker believes a more comprehensive program is necessary. Such a program would be as follows:

1. A comprehensive and wide-spread campaign of educational publicity—one that will reach both the public and the individual mothers.

2. A study of the decrease that has already taken place in the infant death-rate shows that this decrease has occurred in the three groups of contagious, diarrheal and respiratory diseases, in the order given. The group classed as congenital debility shows practically no decline. Here we need the help of the obstetrician, who should not limit the control of his patient to the confinement and the few succeeding weeks, but should insist that the mother place herself under his care and follow his directions during the entire period of pregnancy.

In order to meet and overcome this abnormal death-rate from congenital causes, we must have:

A. Proper education and control of midwives, who in New York city care for over 40 per cent. of the births.

B. Classes for and supervision of pregnant women, using all means to provide them with essential instruction and the means of applying it.

C. A form of insurance that will provide a stated payment for women for a period of at least one month before and one month after confinement, thus obviating the necessity for physical labor on their part during this time.

D. The cooperation of philanthropic forces, relief agencies, and social workers, to provide proper food, hygienic surroundings and freedom from anxiety for the mother during the prenatal period of the child's life.

The socialist will say that the crux of this whole matter is the living wage for the wage earner of the family. To a great extent Baker agrees with him. Such a solution would lighten our labors but we should still have to consider the vast and vexed question of the illegitimate child.

3. The question of institution care versus the placing out system for foundling babies is one that should be practically worked out. The death-rate in these institutions is abnormally high. This rate is greatly reduced when the babies are boarded out and receive that individual attention which every baby needs. Foundling institutions should serve as clearing houses only, furnishing hospital care for the sick babies and immediately placing all others out in properly supervised private homes.

4. Next it is essential that we realize that infant mortality is a year round problem. Misled by the usual increase in summer, we have confined our efforts mainly to that time of the year. The time to save the baby is before it gets sick—not afterward.

Infant Mortality and Morbidity in Berlin in the Summer of 1911

(*Ludwig F. Meyer, Deutsch. med. Wchnschr., Nov. 9, 1911, p. 2090*)

For a solution of the summer mortality of infants Prausnitz has investigated the figures so far obtained. Meyer regards it as especially necessary to supplement these general statistics by individual statistics so that it may be determined whether it is the direct or indirect influence of heat on the organism that is concerned or whether the injury follows from a decomposition of the food. Meyer therefore studied the infants in the orphan asylums of Berlin and also orphans who are cared for outside. They are under constant observation in the so-called convalescent care. These convalescents must be brought to the physician of the asylum every week for examination and exact records are kept. The place in which they are cared for is also inspected every four weeks by a physician, and in the hot months twice weekly by a well-instructed female assistant. There were this year, during the three hot months, June, July and August, 255 children in this convalescent class. The number of nurslings observed in the hospitals amounted to 170 a day. The hospital and outside care supplement each other so that the children may be transferred from one to the other. The temperature during the summer of 1911 was abnormally hot for Berlin. The maximum temperature for June was 85, for July 95 and for August 94 F. There were seven days in June, ten in July and twenty-three in August in which the temperature exceeded 77 F. In the previous year there were fourteen days in June, three in July and only two in August with the same maximum temperature. It is known that summer heat depresses adults leading to diminishing of appetite and a lessened intake of nourishment so that there is a certain condition of hunger or fasting with diminished nutrition. The nurslings, on the other hand, are rather more abundantly fed than usual because of the increased thirst. If the infant only shows a loss in the regular increase in weight this must be explained not in the same way as in the adults but must be considered an injury to the function of nutrition itself. This was shown during the hot months. During July and August, 1911, the average was about one-half of the increase for the corresponding months of the previous year. In July, 1910, it was 590 gm.; in July, 1911, 290 gm.; in August, 1910, 600 gm.; in August, 1911, 350 gm. This is a showing that must be brought into immediate relation with the heat. How far the loss may be accounted for by sweating is unsettled. It is only a step from this depressive action on the general condition to a decided pathogenic action. The health of children is incomparably better in the winter months than in the summer and the types of disease vary distinctly.

	Winter 1911		Summer 1910		Summer 1911	
	Cases	Per cent.	Cases	Per cent.	Cases	Per cent.
Nutritional Diseases	7	17	21	41	32	34
Furunculosis, Abscess, etc. ..	3	6	16	31	26	28
Respiratory Diseases	34	71	12	23	17	18

These figures show first the marked increase in disturbances of nutrition in the hot months and what is certainly not commonly known, a considerable increase in the infections of the skin. Furunculosis, abscess and phlegmon are the immediate results of heat; for by the collection of sweat and the production of decomposition, injuries and inflammations of the skin are produced, which are more serious on account of the decreased resistance occasioned by the heat and the injurious actions of these infections, especially in the young, is reflected in the secondary disturbances of nutrition. One group of diseases noted in 1911 seemed to be due to results of heat stroke. This amounted to 4.2 per cent. of the morbidity, or 1.5 per cent. of the children observed took sick with symptoms similar to heat stroke. Subacute or chronic forms of diarrhea without increase in temperature were lacking in the winter material but are found in the summer proportionally to the prevailing heat. In contrast to the action of heat stroke these disturbances began insidiously and selected ill-nourished children in whom the regular increase in weight had been lacking for weeks previously. During 1910, only rarely was a reduction of the weight observed but in 1911 the reduction went hand in hand with the exaggeration of the entire clinical picture both in the pure alimentary affections as well as in the parenteral affections. Those cases presenting a combination of infectious and alimentary affections had an exceedingly unfavorable course.

Meyer discusses the question as to whether these results are due to decomposition or to diminution of tolerance and says that the insidious nature of the origin of the gastro-intestinal diseases and the aggravation of the clinical picture by heat appear to indicate a diminution of tolerance somewhat in the sense of a diminution of the secretory action of the glands, but this conclusion is not at all definite. Especial care is required in this conclusion as indicated by the observations in the hospitals, where no influence of the heat on the children could be determined, as the increase in weight of the hospital children as in the case of those outside, became smaller. The average monthly increase of all that have been in the institution for fourteen days, including also those in the out-patient service, amounted in June to 400 gm., in July to 320 gm. and in August to 264 gm., and neither overheating, nor increased gastro-intestinal affections, which would indicate an action of heat on the children, were found. As in all institutions the children were loosely clad and their thirst was satisfied by administration of water without any limit. A series of cases of diarrhea occurred but these could be traced to the bad quality of the buttermilk used. Meyer says that putting all of these observations together we must define the influence of heat as follows: The heat acts 1. as an immediate injury to the children by hyperthermia; 2. as a mediate injury by (a) diminution of tolerance, (b) diminution of immunity; (c) by aggravation of the course of the alimentary and infectious diseases; (d) by decomposition of milk.

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